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HEALTH AND ENVIRONMENTAL EFFECTS DOCUMENT FOR BATTERIES--1980

HERAP

Health and Environmental
Risk Analysis Program

ARGONNE
NATIONAL
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HEALTH AND ENVIRONMENTAL EFFECTS DOCUMENT FOR BATTERIES--1980

by the
Division of Environmental Impact Studies
and the
Division of Biological and Medical Research

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prepared for
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ABSTRACT

This Health and Environmental Effects Document (HEED) is an assessment of the ecological and health effects of the near-term electric storage batteries (lead/acid, nickel/zinc, and nickel/iron) as related to electric and hybrid vehicle applications. The storage battery technology is considered in its totality, and emissions are estimated for the complete cycle. For estimating quantities of various emissions, the market penetration is assumed to be 3×10^6 *Pb/acid battery-powered vehicles and* 8×10^6 *each of the Ni/Zn and Ni/Fe battery-powered vehicles on the road by the year 2000.*

Ecological effects are assessed using the Estimated Permissible Concentration (EPC) approach. Pb, S, Cu, Ni, Cd, Zn, and Sb appear to be the most hazardous emission constituents and exceed EPC values for protection of ecology in one or more of the battery-related industries. More definitive, quantitative estimates of damage to the biota and to the ecosystem are not feasible because of paucity of relevant information.

Health effects as a result of increased levels of lead, arsenic, and cadmium in the environment and risks of exposure to arsine, stibine, and antimony trioxide released during charging are assessed. Nickel is recognized as an important metal for health effects analysis and will be addressed in a subsequent HEED. Health effects among the occupationally exposed are also assessed using the toxicological models. Acute and chronic effects of exposure to stibine, arsine, and antimony trioxide are described.

1. EXECUTIVE SUMMARY

1. This Health and Environmental Effects Document (HEED) is an assessment of the ecological and health effects of the near-term electric storage batteries (lead/acid, nickel/zinc, and nickel/iron) as related to electric and hybrid vehicle applications.

2. The storage battery technology is considered in its totality, and emissions are estimated for the complete cycle: (1) mining and milling of the necessary raw materials; (2) manufacture of the batteries and their cases and covers; (3) use of the batteries in electric vehicles, including the charge-discharge cycles; and (4) recycling of spent batteries.

3. For estimating quantities of various emissions, the market penetration is assumed to be 3×10^6 Pb/acid battery-powered vehicles and 8×10^6 each of the Ni/Zn and Ni/Fe battery-powered vehicles on the road by the year 2000. The growth rate for the electric vehicle production is assumed to be 25% per year for the Pb/acid battery and 30% per year each for the Ni/Zn and Ni/Fe batteries.

4. Simple noninteractive dispersion models are used to obtain pollutant levels at various distances up to 30 km from the source and for dispersion over the entire United States.

5. Ecological effects are assessed using the Estimated Permissible Concentration (EPC) approach. Lead, sulfur, copper, nickel, cadmium, zinc, and antimony appear to be the most hazardous emission constituents and exceed EPC values for protection of ecology in one or more of the battery-related industries. More definitive, quantitative estimates of damage to the biota and to the ecosystem are not feasible because of paucity of relevant information.

6. Health effects as a result of increased levels of lead, arsenic, and cadmium in the environment and risks of exposure to arsine, stibine, and antimony trioxide released during charging are assessed. Nickel is recognized as an important metal for health effects analysis and will be addressed in a subsequent HEED. The health effects are assessed on the basis of atmospheric air concentrations. It is recognized that human exposure levels could be higher if foodchain pathways also were considered. However, we believe that atmospheric air concentrations are likely to provide maximum exposure, and contributions from the foodchain pathway may be minimal.

   (a) An individual's probability of developing a blood lead concentration greater than 60 μg/dl, a level indicative of increased potential for central nervous system responses to lead, ranges from 0.0013 to 0.0032, depending on distance from the industrial site (Table 5.1). Upper 95% confidence limits for these estimates indicate an enhanced potential for adverse responses to lead over background levels at distances 5 to 15 km from the sites analyzed. Individual risk corresponding to background levels is found at 50 km from the mine-mill complex. Effects due to dispersal of lead through air at great distances from the site are therefore not anticipated. Significant increases in battery industries are projected by the year 2000 (Table 3.2); increasing numbers of persons could therefore have an increased probability for developing central nervous system responses to lead.

   (b) In the case of arsenic, excess person-years lost as a result of increased lung cancer mortality rates in the vicinity of a secondary lead smelter and a battery manufacturing plant range from 0.23 excess person-years lost (about 84 days) at 5 km to 0.02 excess person-years lost at 30 km from the source (Table 5.2). These values are based on a group of 10,000 persons examined over a ten-year period following 30 years of exposure. The analysis suggests that the increase in risk is negligible from environmental arsenic exposure due to secondary smelting of lead required for increased lead/acid EV battery production.

(c) In the case of cadmium, an increased excess risk of developing renal tubular proteinuria is indicated with increasing cadmium levels associated both by proximity to a lead mine-mill complex and accumulation in the body over time (Table 5.3). At the 5-km distance there is a considerable increase in risk after ten years (1-3 persons per 1000). After a period of 30 years, the excess risk is about 2 per 100, which would imply a significant level of risk. A considerable degree of risk, approximately 2 per 1000, is experienced by those persons at the farthest distance only after a period of 30 years. For those at the middle distances, risk also is most noteworthy at the 30-year point (3-4 per 1000), and is moderate (2-4 per 10,000) at the ten-year point. The excess risk becomes insignificant (defined as 1 per 10^6) at 85 km from the site. Effects due to dispersal of cadmium through air at great distances from the site are therefore not anticipated. Significant increases in mining and milling of lead anticipated by the year 2000 (Table 3.2) could result in local areas of increased exposure to cadmium with accompanying excess risk of developing renal tubular proteinuria.

(d) Health effects among the occupationally exposed are also assessed using the toxicological models. When possible occupational exposure levels were used in conjunction with non-occupational exposures in the dose-response functions, a range of results was seen. The likelihood of having a blood-lead level defined as hazardous was 1 per 100. In a cohort of 10,000 persons followed for ten years (100,000 person-years), 53 person-years could be expected to be lost due to arsenic exposure. Finally, the excess risk of cadmium-induced tubular proteinuria could be about 0.325, using a very conservative definition of the disease.

(e) In an analysis of occupational safety, mean occupational injury and illness rates for persons in the following industries are substantially higher than those reported for persons employed both in other industries related to those required for EV production and in the

entire private sector: battery breaking, secondary lead smelting and refining, storage battery manufacture, and recycling industries for nickel and cobalt (Tables 5.5 and Appendix A, Tables A.2 and A.4). Increased employment in these industries (Tables 3.2, 4.5, and 4.6) could result in a population of persons experiencing a mean rate of occupational injury and illness 1.5- to 2-fold higher than persons with similar skills employed in related industries and approximately 3-fold higher than the mean for the entire private sector.

(f) Acute and chronic effects of exposure to stibine, arsine, and antimony trioxide are described. A potential for reduction in blood hemoglobin levels is identified following chronic low-level exposure to stibine or arsine. Pneumoconiosis with a potential for development of lung tumors is identified as an effect of chronic low-level exposure to antimony trioxide.

2. INTRODUCTION

An initial effort was made to assess the ecological and biomedical effects of commercialization of electric storage batteries (lead/acid, nickel/zinc, and nickel/iron) for near-term electric and hybrid vehicle applications.[1] This follow-up Health and Environmental Effects Document (HEED) contains the emission information in condensed form, contains additional information on dispersion of effluents, and contains an in-depth analysis of the probable health effects resulting from increased levels of lead, arsenic, and cadmium in the environment due to commercialization of battery technology. Risks of exposure to arsine, stibine, and antimony trioxide released during charging also are addressed.

The details of battery technologies necessary for estimation of emissions have been described in the initial document. The storage battery technology was considered in its totality, and emissions were estimated for the complete cycle consisting of (1) mining and milling of the necessary raw materials; (2) manufacture of the batteries and their cases and covers; (3) use of the batteries in electric vehicles, including the charge-discharge cycles; and (4) recycling of spent batteries. Simple noninteractive dispersion models were used to obtain pollutant levels at various distances up to 30 km from the source and for dispersion over the entire United States. These values then were used in the estimation of ecological and human health effects.

After careful scrutiny of the information currently available on commercialization scenarios, we have assumed the market penetration to be 3×10^6 Pb/acid battery-powered vehicles and 8×10^6 each of the Ni/Zn and Ni/Fe battery-powered vehicles on the road by the year 2000. The growth rate for the electric vehicle market was assumed to be 25% per year for the Pb/acid battery and 30% per year each for Ni/Zn and Ni/Fe batteries. These

numbers correspond to the low scenario for the Pb/acid battery and medium scenario for the Ni/Zn and Ni/Fe batteries, as given in References 2 through 7.

The ecosystem effects were evaluated using the Estimated Permissible Concentration (EPC) approach. The health effects were analyzed for lead, arsenic, cadmium, arsine, stibine, and antimony trioxide. Nickel is recognized as an important metal for health effects analysis and will be addressed in a subsequent HEED. The health effects have been assessed on the basis of atmospheric air concentrations alone. It is recognized that exposure levels could be higher if foodchain pathways also were considered. However, budget and schedule have not permitted such additional analysis. Also, we believe that atmospheric air concentrations are likely to provide maximum exposure, and contributions from the foodchain route may be minimal. The occupational injury and illness rates for industries related to battery technology also were examined and effects due to increased employment in the battery industries have been assessed.

The health and environmental effects resulting from operation of power plants to charge the batteries were not considered in this HEED. A more thorough analysis would involve consideration of the tradeoffs of discontinuation of gasoline usage and production of electricity to charge the batteries. Such an analysis may be attempted in future HEEDs.

References for Section 2

1. R.K. Sharma et al. "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," Argonne National Laboratory, ANL/ES-90, Argonne, IL, 1980.

2. M. Millar "EHV Programmatic Environmental Assessment, Discussion Draft #1, Projection of Bus Sales, Vehicle Population and VMT to the Year 2000," Transportation Energy Systems, Energy and Environmental Systems Division, Argonne National Laboratory, Argonne, IL, October 1978.

3. R.E. Knorr and P.D. Patterson. "EHV Programmatic Environmental Assessment, Discussion Draft #2, Projection of Light Truck Stock to the Year 2000," Transportation Energy Systems, Energy and Environmental Systems Division, Argonne National Laboratory, Argonne, IL, December 1978.

4. M. Millar, "EHV Programmatic Environmental Assessment, Discussion Draft #3, Projection of Automobile Stock to the Year 2000," Transportation Energy Systems, Energy and Environmental Systems Divison, Argonne National Laboratory, Argonne, IL, February 1979.

5. M.J. Bernard, III, "EHV Programmatic Environmental Assessment, Discussion Draft #6 (and #6 Addendum), Federal EV Investment Scenarios and EV Markets," Transportation Energy Systems, Energy and Environmental Systems Division, Argonne National Laboratory, Argonne, IL, December 1978.

6. M.J. Bernard, III, "EHV Programmatic Environmental Assessment, Discussion Draft #7, Regional Variation in EV Market Penetration," Transportation Energy Systems, Energy and Environmental Systems Division, Argonne National Laboratory, Argonne, IL, January 1979.

7. M.J. Bernard, III, "EHV Programmatic Environmental Assessment, Discussion Draft #11, EHV, VMT and Direct Energy Use by Vehicle Type, Scenario and Federal Region," Transportation Energy Systems, Energy and Environmental Systems Division, Argonne National Laboratory, Argonne, IL, June 1979.

3. BATTERY CYCLES

3.1 THE LEAD/ACID BATTERY

3.1.1 Cycle Flow Rates and Emission Rates

The main stages in the lead/acid battery cycle consist of primary production of new battery materials, manufacture of batteries, battery use, battery breaking, and secondary recovery of used materials. Primary production of lead is further separated into lead mining and milling, and smelting and refining. The waste effluents from each stage are categorized into those emitted into the atmosphere, into water, and solid wastes.

The flow rates of effluents emitted at each stage depend on the level of industrial activity associated with each stage, as well as on the pollution control techniques used and on other factors. In particular, the following two assumptions are used here:[1]

- The assumed scenario[2] is that lead/acid battery use will grow at a rate of 25% per year to reach a fleet size of 3×10^6 lead/acid electric vehicles by the year 2000, with a battery-use lifetime of 3.3 years.

- At each stage in the cycle all materials are produced in plants using modern pollution control techniques even though at present only a few plants may be using some of these procedures.

These assumptions and data from the literature enable one to calculate material flow rates in the cycle and pollutant emmission rates from each stage in the cycle for any scenario year. Further details are given in Sharma et al.[1] In Table 3.1, total lead flow rates between cycle stages are given in MT/day, and air, water, and solid emission rates of Pb, As, Cd, and Ni are given in kg/day from the cycle stages for the scenario year 2000. The total

Table 3.1. Lead Flow Rates and Air, Water, and Solids Emission Rates of Pb, As, Cd and Ni from the Stages in the Lead/Acid Battery Cycle for the Scenario Year 2000

Stages and Lead Flow Rates in the Battery Cycle	Air Emissions (kg/day)				Water Emissions (kg/day)				Solid Emissions (kg/day)			
	Pb	As	Cd	Ni	Pb	As	Cd	Ni	Pb	As	Cd	Ni
Lead Mining Milling	3,300	-	9.	13.	18.	-	0.82	-	1.6×10^4[b]	-	280.[b]	1,100[b]
1100 MT/day												
Primary Lead Smelting Refining	1,300	0.87[c]	3.7[c]	-	0.43	0.14	0.43	0.16	1.2×10^4	-	600.	-
1100 MT/day												
Lead/Acid Battery Mfg.	100	0.0026	-	-	22.	0.0075	-	-	750.[d]	0.26[d]	-	-
1800 MT/day												
Battery Use	[See text]				0	0	0	0	0	0	0	0
700 MT/day												
Battery Breaking	-	-	-	-	0.11	0.0072	0.0072	0.037	23.	0.80	0.68	0.18
700 MT/day												
Secondary Lead Smelting Refining	900	0.32	-	-	0	0	0	0	140.	0.040	-	-
700 MT/day (recycle)												
Total Emissions (kg/day)	5,600	1.2	13.	13.	40.	0.15	0.44	0.20	2.9×10^4[d]	1.1	880	1,100

[a] For details see Ref. 1, Sec. 2. The entries with dashes indicate that insufficient information was available to estimate the emission rate. The zeros mean no emissions.

[b] The entries refer to the content of mill tailings

[c] These values were obtained from fugitive Pb, As, and Cd emissions from a Missouri Smelter [P. Constant, M. Marcus, W. Maxwell, Sample Fugitive Lead Emissions from Two Primary Lead Smelters, Midwest Research Institute, Kansas City Mo. 1977. Report No PB-276-356] by multiplication by the ratio of total to fugitive Pb emissions given in Table 2.8 of Ref. 1.

[d] These values refer to the wet process only.

emission of each type of pollutant summed over all the cycle stages also is given.

Air emissions from battery use are gases, which are produced, for the most part, during charging. Besides H_2, O_2, small amounts of H_2SO_4 mist, the toxic gases arsine (AsH_3) and stibine (SbH_3) also are produced. The production rate of these gases is quite dependent on charge parameters.

The AsH_3 and SbH_3 emissions expected from battery use are listed in Table 3.2. The data are based on a study of emissions from a 21-kWh lead/acid battery-powered van.[3] The amounts of antimony trioxide (Sb_2O_3) listed are calculated on the basis of the assumptions that SbH_3 has a 9-minute half-life (which is the average of the reported literature values of 6 to 12 minutes)[4] and that all SbH_3 coverts to Sb_2O_3 before leaving the garage. The discharge measurements were obtained while the van was cruising at 40 km/hr.[3] The production rate and total production columns of the table are obtained from AsH_3 and SbH_3 battery cell off-gas concentrations. The values for the total emissions from 3×10^6 EVs are calculated assuming one charge-discharge cycle every 2.4 days,[1] and one equalization charge after four regular charges.[5] The AsH_3 and SbH_3 concentrations given in Table 3.2 are time-averaged concentrations. However, it is known that most of the AsH_3 and SbH_3 is produced in the overcharge portion of the charge.[5] Thus, during the time in which overcharge is occurring, AsH_3 and SbH_3 concentrations would be larger than the table entries for regular charging by a factor equal to the ratio of the total charge time to the overcharge time. For an overcharge time of three hours and a total charge time of ten hours, the factor is 3.3.

It also should be noted that significant residual SbH_3 and AsH_3 concentrations were measured in the van during discharge,[3] although these gases were generated during charging. Concentrations measured at the battery compartment exhaust were much higher than those shown for the inside of the van.[3] These data indicate the need for ventilation of the battery compartment during discharge, as well as during charge.

Significant amounts of Sb_2O_3 also are produced during charge and during discharge as a result of the SbH_3 reacting with the oxygen in the atmosphere.

Table 3.2. Air Emissions from Charge and Discharge of Lead/Acid Battery Powered Van.[a]

Chemical Species	One Regular Charge					
	Production Rate,[b] mg/min.	Time, min.	Total Production, mg	Time-Averaged Concentration, mg/m³		
				Garage[c]		Van[c]
				Calculated	Measured	Measured
AsH_3	0.34	600	20	0.2[d]	0.0036-0.011	0.0034-0.0048
SbH_3	0.42	600	250	0.054[e]	0.010-0.086	0.008-0.041
Sb_2O_3[f]	0.45	600	270	270 mg	240 mg[g]	6.5 mg[g]

Chemical Species	One Equalization Charge					
	Production Rate,[b] mg/min.	Time, min.	Total Production, mg	Time-Averaged Concentration, mg/m³		
				Garage[c]		Van[c]
				Calculated	Measured	Measured
AsH_3	0.0035	240	0.84	0.0084[d]	0.0032	0.0022[h]
SbH_3	0.31	240	74	0.040[e]	0.034	0.006[h]
Sb_2O_3	0.34	240	82	82 mg	70 mg	0.61 mg[h]

Chemical Species	One Discharge			
	Production Rate,[b] mg/min	Time, min.	Total Production, mg	Time-Averaged Concentration Measured in van,[c] mg/m³
AsH_3	0.0044	40	0.18	0.021[h]
SbH_3	0.026	40	1.0	0.02[h]
Sb_2O_3	0.027	40	1.1	0.33 mg[h]

Total Emissions from 3×10^6 EVs, kg/day[i]	
AsH_3	26
SbH_3	340
Sb_2O_3	370

[a] Data is from Reference 3. Battery size is 21 kWh. During discharge the van was cruising at about 40 km/hr.

[b] Calculated from cell outlet data of Table 1 in Reference 3.

[c] Assumed garage volume = 100 m^3, van volume = 5.5 m^3 (Refs. 1 and 3).

[d] Values are concentrations at end of charge, AsH_3 is stable.

[e] Values are calculated from production rate assuming that $SbH_3 \rightarrow Sb_2O_3$ with a half-life = 9 min. (Ref. 4) and steady state conditions exist for duration of charge. Decay of SbH_3 inside the battery is neglected.

[f] Amounts are calculated under assumption that all SbH_3 decays before leaving van or garage.

[g] Values are calculated from average of measured concentration assuming steady state conditions.

[h] These values are upper limits.

[i] Values are calculated assuming one charge-discharge cycle every 2.4 days and one equalization charge after four regular charges.

It is difficult to predict what happens to this material and what concentrations of Sb_2O_3 dust humans are exposed to in vehicles and garages. As a very crude first step it will be assumed that the particle size of Sb_2O_3 is such (0.1-0.3 μm) that no gravitational settling occurs and that deposition by turbulent diffusion is slow, with a half-life of at least days.[6] In this case the Sb_2O_3 garage concentration and in-vehicle concentration at the end of a regular charge are 2.4 mg/m^3 and 1.2 mg/m^3 respectively. Opening the garage and driving out the vehicle will remove much of this accumulated material to the outside by turbulent diffusion. A similar removal will occur when a person enters the vehicle. However, a person working in a closed garage at the end and after the termination of charging would be exposed to the full concentration.

While driving or riding in the vehicle, one is exposed to some Sb_2O_3 resulting from the prior charge as well as the production during discharge. The average concentration of Sb_2O_3 in a closed vehicle during a 40-minute trip is 0.030 mg Sb_2O_3/m^3 (from Table 3.2). This concentration average was calculated on the basis of a constant production rate, and is lower for shorter driving times. Also, driving with air conditioning or an open window will greatly reduce exposure.

Details of the health effects of AsH_3, SbH_3 and Sb_2O_3 are discussed in Section 5.

3.1.2 Dispersion of Effluents

Atmospheric dispersion and deposition are important pathways for dispersing pollutants in the environment since pollutants can be transported easily over long distances and washed out by precipitation or settle and/or diffuse into soil and water. A simple Gaussian dispersion analysis (see Ref. 1, Sec. 5 for details) of the air emissions from the various point sources in the lead/acid battery cycle leads to the results portrayed in Figures 3.1 and 3.2. The ground-level atmospheric concentrations of lead, arsenic, and cadmium as a function of distance from the source are shown in Figure 3.1 for several industries in the lead/acid battery cycle. Curves for nickel,

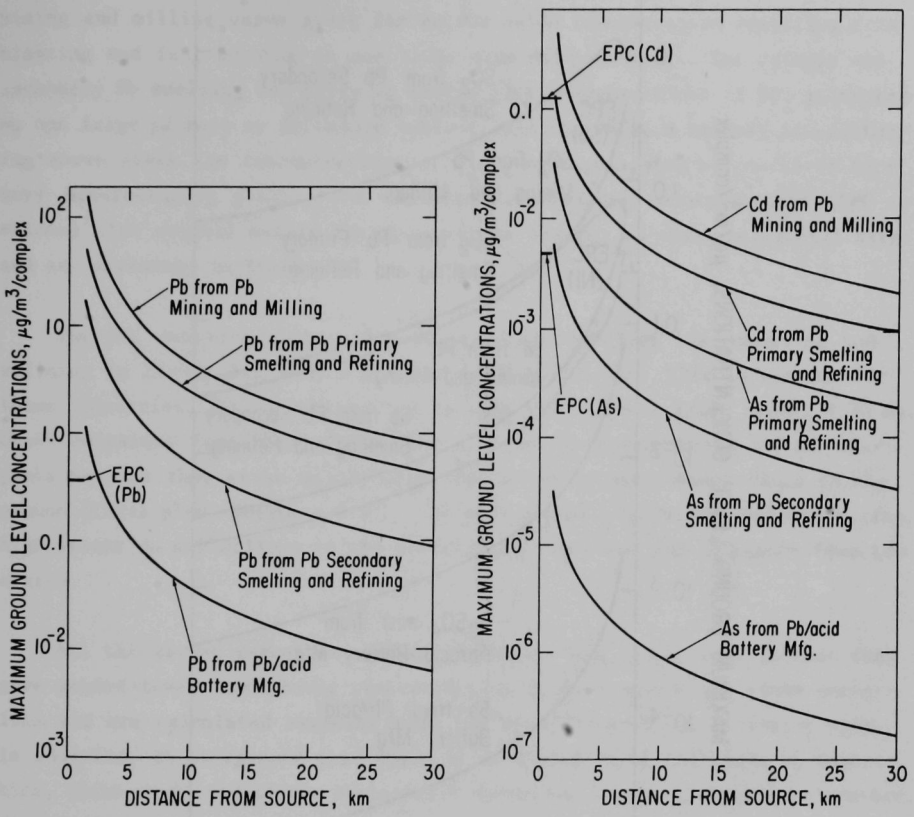

Fig. 3.1. Ground-Level Atmospheric Concentrations of Pb, As and Cd under the Plume Centerline from Sources in the Lead/Acid Battery Cycle. Each curve is labelled as to the effluent (Pb, As or Cd) and the industry stage to which it refers, and gives the concentrations as a function of distance from one standard plant or complex. Estimated Permissible Concentrations of Pb, As and Cd are given as short horizontal bars on the ordinate.

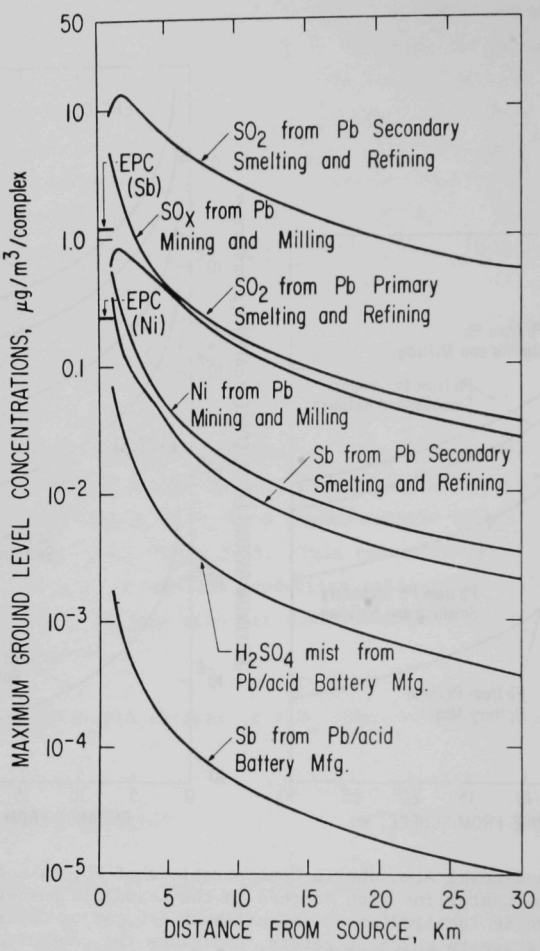

Fig. 3.2. Ground-Level Atmospheric Concentrations of Ni, Sb and SO_x or H_2SO_4 Mist under the Plume Centerline from Sources in the Lead/Acid Battery Cycle. Each curve is labelled as to the effluent and the industry stage to which it refers, and gives the concentrations as a function of distance from one standard plant or complex. Estimated Permissible Concentrations of Ni and Sb are given as short horizontal bars on the ordinate.

antimony, and sulfur oxides or H_2SO_4 mist are given in Figure 3.2. Source air emissions were obtained from Ref. 1 and Table 3.1. In Figure 3.2, the Pb mining and milling curve gives the sulfur oxide concentration resulting from blasting and fuel burning in one large mine-mill complex. The primary and secondary Pb smelting and refining curves give concentrations of SO_2 generated by one large primary or secondary smelter, and the Pb/acid battery manufacturing curve gives the concentrations of H_2SO_4 mist generated by one large battery manufacturing plant. The estimated permissible concentrations (EPC values)[7] for several metals are given on the figures as short horizontal bars and are discussed in Section 4.

The SO_2 emission curves from primary and secondary lead smelting and refining in Figure 3.2 show a peak at small distances. This is because for these industries, SO_2 is emitted from a high stack (level plume height = 70 m). Other emissions from these industries or from other industries do not show peaks because they arise mainly from fugitive or other sources close to the ground (level plume height = 5 m). The peak occurs because the emissions from high stacks do not diffuse to the ground until they are some distance from the source.

All the curves represent maximum, or worst-case, conditions in that they give ground-level atmospheric concentrations directly under the plume centerline and are calculated assuming that the wind blows in one direction only. In addition, it is assumed that there is no depletion of the plume by deposition, which would reduce the atmospheric concentrations, especially at greater distances.

The curves in Figures 3.1 and 3.2 can be used to calculate worst-case deposition rates of effluents by assuming that at each point the deposition rate is proportional to the ground-level atmospheric concentration and that all deposition occurs within 50 km downwind of the source. The values are obtained by integration of the curves in Figures 3.1 and 3.2 over appropriate distance intervals and normalizing according to the above assumption.

The maximum deposition rates under the plume centerline are given in Figure 3.3 for lead, arsenic, and cadmium and in Figure 3.4 for nickel,

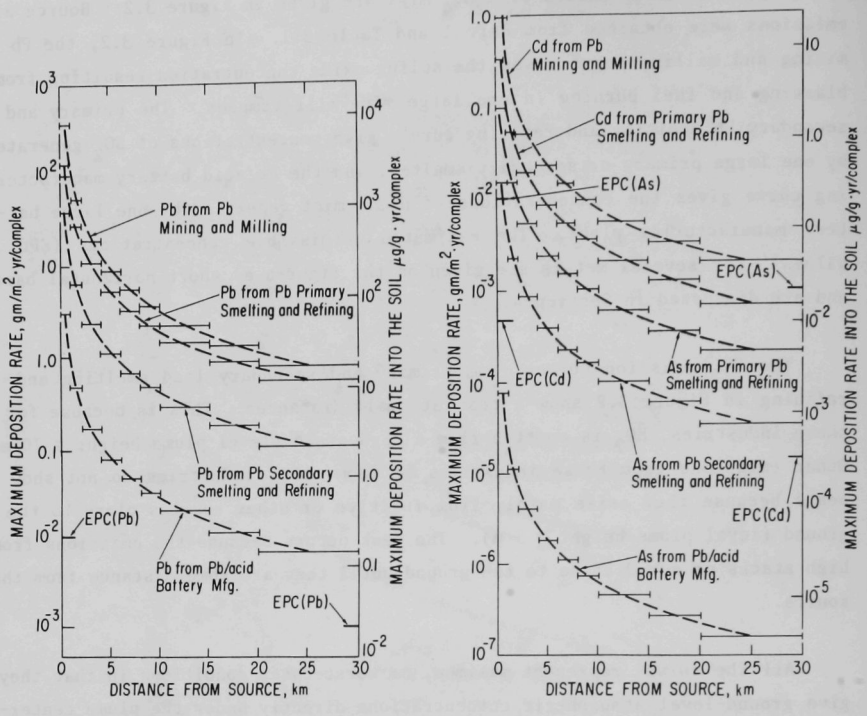

Fig. 3.3. Maximum Deposition Rates of Pb, As and Cd under the Plume Centerline from Sources in the Lead/Acid Battery Cycle. Each horizontal line segment represents the rate of deposition into a narrow region under the plume. The short vertical bars on the segments give the distance interval for the region. All segments which give deposition rates for a given effluent and industry source are connected by a dashed curve. The left and right hand ordinate scales denote the rates of deposition onto the surface in $g/m^2/yr$ and into the soil in $\mu g/g/yr$. The curves are all normalized to the source emission rate of a large standard plant or complex. Estimated Permissible Concentrations for Pb, As and Cd in soil and water are given as short horizontal bars on the respective right or left hand ordinates. Further details are given in the text.

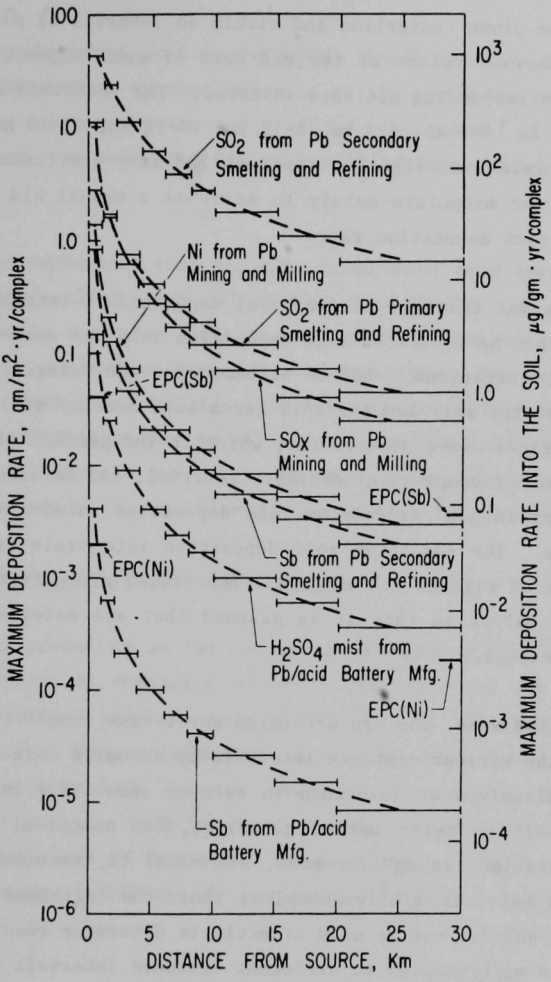

Fig. 3.4. Maximum Deposition Rates of Ni, Sb and SO_x or H_2SO_4 Mist under the Plume Centerline from Sources in the Lead/Acid Battery Cycle. See caption of Fig. 3.4 and text for details.

antimony, and SO_2-H_2SO_4 mist from various sources in the lead/acid battery cycle. Each line segment with end bars represents, for one large factory or complex as the source, the average deposition rate in $g/m^2 \cdot yr$ in an area close to and under the plume centerline and within an interval of distance from the source. The abscissa values of the end bars of each segment give the end-points of the corresponding distance interval. The intervals selected are 0-1 km, 1-2 km, 2-4 km, 4-6 km, 6-8 km, 8-10 km, 10-15 km, 15-20 km, and 20-30 km. The deposition rate line segments for each industry are connected by dashed curves through the midpoints mainly to serve as a visual aid in identifying the source for each deposition rate.

If one assumes that all the material deposited is retained in the soil, the rates can then be converted into deposition into the soil or annual incremental soil concentrations. If one assumes that the material is retained in the top 3 cm of the soil and the soil has a bulk density of 1.5 g/cm^3,[8] then one fixed numerical conversion factor, which is independent of effluent, type of source, plant or complex, or distance interval, can be used to convert the deposition rates in $g/m^2/yr/complex$ into deposition rates into the soil in $\mu g/g/yr/complex$. The resulting soil deposition rate scale is shown on the right ordinates of Figures 3.3 and 3.4. The values given by the line segments are worst-case values in that it is assumed that all material deposited is retained by the soil.

In a similar vein, one can determine worst-case runoff concentrations of effluents in the various distance intervals by assuming that all the material deposited is dissolved or suspended in rain or snow. For an average annual precipitation of one meter water equivalent, the numerical values of the runoff concentration, in mg/L/complex, are equal to the numerical values of the deposition rate, in $g/m^2/yr/complex$; thus, the left-hand ordinate scales of Figures 3.3 and 3.4 can be used directly to determine runoff concentrations of effluents in mg/L/complex at different distance intervals from the sources.

EPC concentrations[7] for the soil are given for various elements as short horizontal lines on the right-hand ordinates of the tables. Each value is marked with the particular effluent to which it refers. Corresponding water EPC concentrations[7] (mg/L) are given as lines on the left ordinate, where the

equality noted above between the runoff concentrations (mg/L) and deposition rates ($g/m^2/yr$) has been used. These EPC values are discussed in detail in Section 4.

Under the assumptions used to calculate runoff concentrations, the concentration is independent of what fraction of the precipitation runs off and what fraction is retained by the soil. It will be assumed in later sections that one-fourth of the precipitation runs off and the remainder is retained by the soil.

It should be emphasized that the dispersion model used here contains many simplifying approximations. Removal of these and use of wind velocity profiles appropriate to a source location may lower effluent concentrations and deposition rates by as much as two orders ot magnitude. How much these values would be lowered depends on conditions in the vicinity of the complex. On the other hand, the effluent-source emission rates are based on plants employing modern control technology. Any plants applying older control technology or very few controls would release more effluents than those calculated here.

3.1.3 Effluent Dispersion at Large Distances

The material presented so far is appropriate for study of health and environmental effects at distances at which diffusion has not washed out vertical and horizontal concentration gradients. To calculate the total effect of a pollutant over a large area (e.g., the whole United States) produced by the Pb/acid battery industry operating at scenario levels, a different simplified analysis that treats concentrations and deposition effects at large source distances is required. In particular, it is assumed here that the concentration of a pollutant at a distance "r" from a source, "$C_{UH}(r)$," is given by

$$C_{UH}(r) = \frac{Q}{2\pi URH} e^{-\lambda r} . \qquad (1)$$

Here U is the wind speed, Q is the source pollutant emission rate, H is the mixing height, and λ is the pollutant decay length given by

$$\lambda = \frac{0.693}{Ut_{1/2}} , \tag{2}$$

where $t_{1/2}$ is the deposition half-life of the pollutant.

The usefullness of Equation 1 depends on the following assumptions:

- The vertical concentration is independent of height up to the mixing height.
- Loss of material by deposition affects the whole vertical column uniformly and not just concentrations close to the ground.
- The population density is uniform over the large area of concern.

Under the uniform population density assumption, $C_{UH}(r)$ given by Equation 1 represents a reasonable average over wind directions, which is independent of the wind direction frequency function.

A simplified calculation of the health effects of a pollutant on a large population can be carried out as follows: under the linear no-threshold assumption (at least over concentration ranges of interest) the probability per person at position r, θ that a specific health effect E occurs due to inhaling pollutant I at concentration $C(r,\theta)$ is given by:

$$P_E^I(r,\theta) = K_E^I C(r,\theta) . \tag{3}$$

Here polar coordinates are used and the possibility that the concentration and thus the probability is r and θ dependent is included. If $d(r,\theta)$ is the

population density, then the total number of people with health effect E due to pollutant I in the area A is given by:

$$N_E^I = \int_A d(r,\theta) P_E^I(r,\theta) r dr d\theta \ . \qquad (4)$$

In the situation of interest $d(r,\theta) = d$ independent of r and θ. Also, because of the exponential deposition one can extend A to infinity without appreciable error provided that the radius of A is at least as large as λ^{-1}. In this case use of Equations 1, 3 and 4 gives:

$$N_E^I = \frac{K_E^I Q_I d}{2\pi UH} \int_0^{2\pi} d\theta \int_0^{\infty} \frac{e^{-\lambda r}}{r} \cdot r dr \ . \qquad (5)$$

Integration and use of Equation 2 gives the final results:

$$N_E^I = \frac{K_E^I Q_I dt_{1/2}}{0.693H} = K_E^I \bar{C}^I \ , \qquad (6)$$

where $\bar{C}^I$ is an average person (pollutant-I) concentration.

The values of $\bar{C}^I$ for the scenario year 2000 are given in Table 3.3 for the pollutants listed in Table 3.1. The value for d was taken to be 23 people/km^2, the average density of people in the United States; H = 740 m is given by $1/H = 1/2\ (1/H_M + 1/H_A)$, where H_M = 500 m and H_A = 1400 m, the respective morning and afternoon average mixing heights.[9] A crude estimate $t_{1/2}$ = 2 days is used along with the values of Q_I given by the total air emissions from all stages in the Pb/acid battery cycle in Table 3.1. (Since the average wind speed over the United States is about 5 m/s,[9] $\lambda^{-1} \approx$ 1200 km, which is roughly the radius of the United States.)

The values in Table 3.3 were derived assuming that all emission sources can be replaced by one very large point source. Such an assumption is admittedly crude. However, it is approximately true for primary lead production and is not unreasonable for large distances from the source.

Table 3.3. Average Person-Pollutant Concentrations in the Scenario Year 2000 from All Stages in the Lead/Acid Battery Cycle (μg-persons/m^3)

Pollutant	Pb	As	Cd	Ni
$\bar{C}^I$	5.0×10^4	11	120	120

The values given in tables pertain to a scenario with a 25% per year growth rate. For a scenario with 3×10^6 Pb/acid EVs with zero growth rate, the values in Tables 3.1 and 3.3 would be different. In particular, primary lead production values drop to 10% of the values in the tables (primary production then makes up for cycle losses only), battery manufacturing drops by 33%, and battery use, battery breaking, and secondary recovery values all increase by about 50% over the values in the tables.

3.2 THE NICKEL/ZINC BATTERY

3.2.1 Cycle Flow Rate and Emission Rates

The scenario used here predicts that the number of Ni/Zn EVs in use will grow at a rate of 30% per year and result in 8×10^6 EVs by the year 2000.[2] Ni/Zn EVs with 25-kWh batteries are projected to have an average range of 250 km and a battery lifetime of 9.4 years (see Ref. 1, Sec. 3). Both values are larger than the corresponding values for lead/acid batteries and are indicative of the attractiveness of Ni/Zn batteries for EV use provided the technological problems can be overcome.

Projecting the level of industrial activity and resulting emissions from industries producing and recycling Ni/Zn batteries is more speculative than for the lead/acid batteries because there is no existing large-scale industry producing these batteries. The small amount of data available on the Ni/Cd battery industry is only of limited use because the industry is small compared to the lead/acid battery industry.[10] Thus, projected flow rates and emission

rates are even more tentative than for the lead/acid battery cycle. Following the development used for Table 3.1 and using available estimates, one can generate some material flow rates and pollutant emission rates for some stages in the Ni/Zn battery cycle. The results are given in Table 3.4, along with total emission rates for each pollutant summed over the stages. The large number of blanks in the table indicate the lack of information available on which to base estimates. In particular, emissions for recycling and recovery are not given because quantitative estimates would be too speculative.

The emission factors given in Table 3.4 for many of the industrial stages in the Ni/Zn battery cycle are, at best, rough estimates. Almost all nickel mining, milling, smelting, and refining goes on outside the United States, so it is difficult to obtain emission information for these industries. Other than a few Ni/Zn batteries made for research or demonstration purposes, there is no Ni/Zn battery manufacturing or recycling industry. The existing nickel-cadmium battery manufacturing industry is too small and specialized; nevertheless, the water Ni emission factor given in Table 3.4 for battery manufacture is based on a study of this industry.[10]

The emissions from use of batteries in EVs are gases, chiefly H_2 and O_2, given off during recharging. As was the case for Pb/acid batteries, these emissions occur during recharging or during regenerative breaking. No emissions other than a small amount of KOH mist are expected during use.

3.2.2 Dispersion of Effluents

Atmospheric dispersion of effluents from one large plant or complex can be obtained by the same methods outlined for the Pb/acid batteries and are given in more detail in Ref. 1. Concentrations for Ni, Cd, and Co are given in Figure 3.5, and for Pb, As, Sb, and SO_2 in Figure 3.6. Both the effluents and their sources are labeled for each curve. The values shown in the curve for Ni from Ni smelting and refining have been multiplied by 0.1 to make presentation easier. The EPC values[7] are given as short horizontal lines on the ordinate and will be discussed later.

Table 3.4. Materials Flow Rates and Air, Water, and Solids Emission Flow Rates of Ni, Pb, Cd, and As from Stages in the Ni/Zn Battery Cycle for the Scenario Year 2000[a]

Stages and Material Flow Rates in the Battery Cycle	Air Emissions (kg/day)				Water Emissions (kg/day)				Solid Emissions (kg/day)			
	Ni	Pb	Cd	As	Ni	Pb	Cd	As	Ni	Pb	Cd	As
Zinc Mining Milling	0.04	12.	0.04	-	-	20.	4.	-	1000	1.5×10^4	260.	-
410 MT/day												
Primary Zinc Smelting Refining	-	0.66	5.	0.14	-	1.8	1.8	0.36	-	19.	26.	-
410 MT/Day												
Primary Nickel Smelting Refining	1.9×10^3	160.	-	-	160.	-	-	-	-	-	-	-
780 MT/day												
KOH Mfg	-	-	-	-	-	0.008	-	-	-	16.	-	-
280 MT/day												
Ni/Zn Battery Manufacture	960.	-	-	-	470.	-	-	-	-	-	-	-
Battery Use Recovery	-	-	-	-	-	-	-	-	-	-	-	-
Total Emissions (kg/day)	2.8×10^3	170	5.	0.14	630.	22.	5.8	0.36	1000	1.5×10^4	290.	-

[a]For details see Section 3 of Ref. 1. The dashes indicate that insufficient information was available to estimate emission factors.

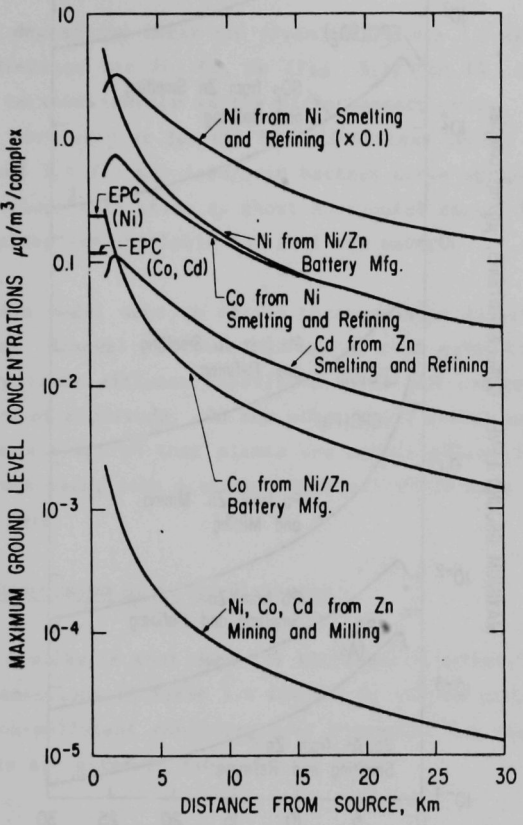

Fig. 3.5. Ground-Level Atmospheric Concentrations of Ni, Cd and Co under the Plume Centerline from Sources in the Ni/Zn Battery Cycle. See caption of Fig. 3.1 and text for details.

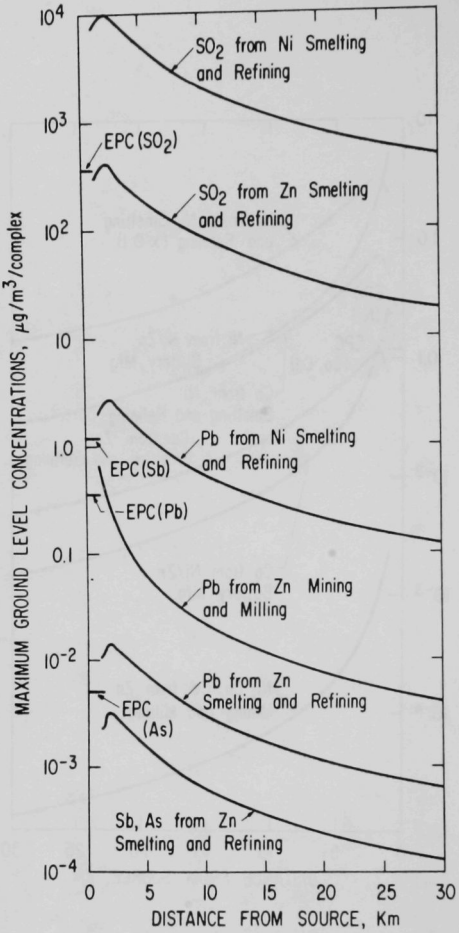

Fig. 3.6. Ground-Level Atmospheric Concentrations of Pb, As, Sb and SO_2 under the Plume Centerline from Sources in the Ni/Zn Battery Cycle. See caption of Fig. 3.1 and text for details.

The emission curves for Ni and Zn smelting and refining pertain to stack emissions only (70-m-level plume height) and include no emission sources close to the ground. Thus, these curves show a peak at small distances, just as do the SO_2 curves for lead smelting and refining in Figure 3.2.

The maximum deposition rates are given in Figures 3.7 and 3.8 as a function of source distance for Ni, Cd, Co (Fig. 3.7) and Pb, As, Cd, and SO_2 (Fig. 3.8) from various sources in the Ni/Zn battery cycle. The same worst-case assumptions were used as for the Pb/acid battery cycle. The description of Figures 3.3 and 3.4 for the lead/acid battery cycle applies to these figures also. EPC values[7] are given as short horizontal bars. None is given for SO_2 because no values are available for soil and water.

The dispersion model used to obtain these results contains many simplifying assumptions. Removal of these and use of more realistic wind velocity distributions may lower effluent deposition rates and concentrations by as much as two orders of magnitude. On the other hand, source emission rates are calculated on the assumption that plants use modern pollution control technology. Any plants using less stringent controls would have larger emissions than those given here.

3.2.3 Effluent Dispersion at Large Distances

An analysis similar to that done for the Pb/acid battery (Table 3.3), but using total air emissions in Table 3.4 for the Q_I values in Equation 6, gives the average person-pollutant concentrations projected for the scenario year 2000. The results are given in Table 3.5.

As is the case for the entries in Table 3.3, the results shown in Table 3.5 can be used to estimate large-scale health effects due to the industrial support of a fleet of 8×10^6 Ni/Zn EVs in the scenario year 2000. Because of the 30% growth rate and 9.4-year battery lifetime, the contributions to these values from battery recycling would be small. However, a different scenario of a fleet of 8×10^6 Ni/Zn EVs with zero growth rate and essentially complete recycling of the Ni and Zn, would require Ni and Zn flow rates in secondary recovery operations about one-third the primary flow rates given in Table 3.4.

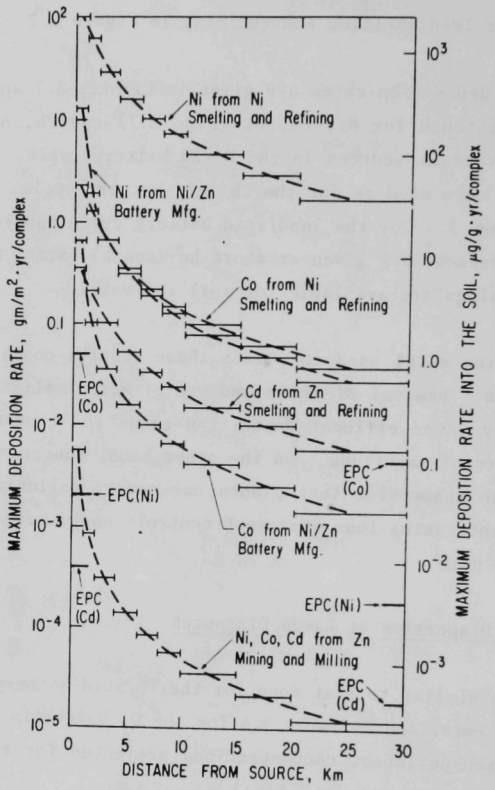

Fig. 3.7. Maximum Deposition Rates of Ni, Cd and Co under the Plume Centerline from Sources in the Ni/Zn Battery Cycle. See caption of Fig. 3.3 and text for details.

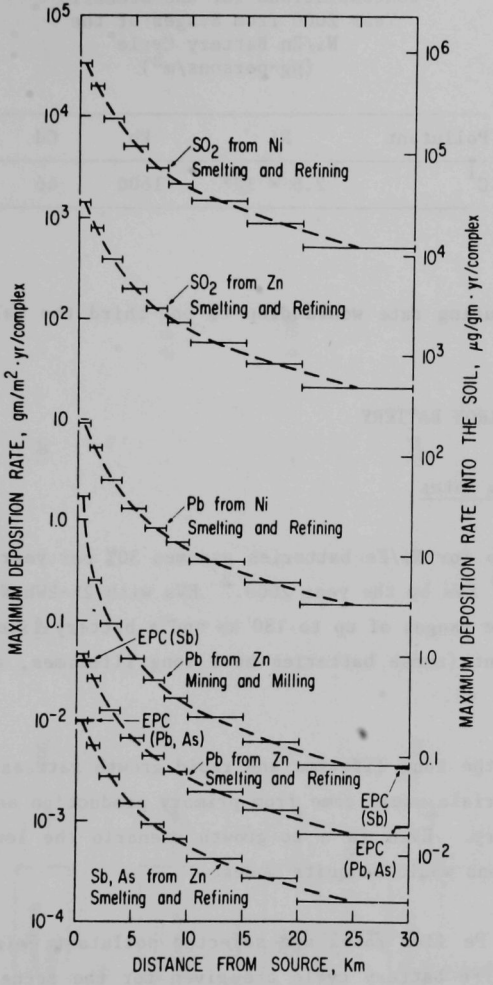

Fig. 3.8. Maximum Deposition Rates of Pb, As, Sb and SO_2 under the Plume Centerline from Sources in the Ni/Zn Battery Cycle. See caption of Fig. 3.3 and text for details.

Table 3.5. Average Person-Pollutant Concentrations for the Scenario Year 2000 from Stages of the Ni/Zn Battery Cycle (μg-persons/m^3)

Pollutant	Ni	Pb	Cd	As
$\bar{C}^I$	2.6×10^4	1600	46	1.3

The KOH manufacturing rate would drop to one-third the value given in the table.

3.3 THE NICKEL/IRON BATTERY

3.3.1 Cycle Flow Rates

The scenario for Ni/Fe batteries assumes 30% per year growth to reach a total of 8×10^6 EVs by the year 2000.[2] EVs with 25-kWh Ni/Fe batteries are projected to have ranges of up to 180 km and a battery lifetime of 22.5 years before replacement (these batteries have long lifetimes, about 200 cycles) (see Ref. 1, Sec. 4).

Because of the long lifetime and rapid growth rate assumed, essentially all battery materials would come from primary production and almost none from secondary recovery. Even in a no-growth scenario the level of secondary recovery operations would be quite modest.

The Ni and Fe flow rates and selected pollutant emission rates from stages in the Ni/Fe battery cycle are given for the scenario year 2000 in Table 3.6. Total pollutant emission rates summed over the cycle stages also are given. As was the case for Table 3.4, dashes mean that insufficient information was available to make an estimate. Emissions for recycling and recovery are not given because quantitative estimates would be too speculative. In any case, because of the long battery lifetime, emissions would be expected to be small.

Table 3.6. Materials Flow Rates and Air, Water, and Solids Emission Flow Rates of Ni and Pb from Stages in the Ni/Fe Battery Cycle for the Scenario Year 2000[a]

Stages and Material Flow Rates in the Battery Cycle	Emissions (kg/day)					
	Air		Water		Solid	
	Ni	Pb	Ni	Pb	Ni	Pb
Nickel Smelting Refining	1200	100	100	-	-	-
490 MT/day						
Iron, Steel Production	0.04	4	-	0.8	-	-
1200 MT/day						
KOH Mfg.	-	-	-	0.006	-	-
220 MT/day						
Ni/Fe Battery Mfg.	1000	-	400	-	-	-
Battery Use Recovery	-	-	-	-	-	-
Total Emissions	2200	100	500	0.8	-	-

[a]For details, see Ref. 1, Sec. 4.

3.3.2 Dispersion of Effluents

The dispersion of the emissions into the atmosphere generated by one large plant or complex and the deposition rates of the emissions are calculated from the data in the same manner as was done for Ni/Zn and Pb/acid batteries. No curves are given here for the effluents of Ni/Fe batteries because they are essentially the same as those discussed in Section 3.2 for Ni/Zn batteries. Since Ni smelting and refining is common to both the Ni/Zn and Ni/Fe battery industries, the data for atmospheric dispersion and deposition of Ni, Pb, Co, and SO_2 from Ni smelting and refining are given directly by the corresponding curves in Figures 3.5 through 3.8. Examination of the appropriate entries of Tables 3.4 and 3.6 shows that the rate of Ni air emissions from one Ni/Fe battery manufacturing plant is the same as from one Ni/Zn battery manufacturing plant. Thus, the Ni emission and deposition curves given in Figures 3.5 and 3.7 also apply to Ni/Fe battery manufacturing plants.

Dispersion curves are not given for iron and steel manufacturing because the amounts needed for the Ni/Fe batteries in a large fleet are quite small compared with the total production. Thus, the impact of a large Ni/Fe battery industry on the steel industry in terms of the amount of steel and iron needed in the battery is small and is omitted here.

3.3.3 Dispersion of Effluents at Large Distances

The average person-pollutant concentrations projected for the scenario year 2000 have been calculated from the values given in Table 3.4 by the method used to obtain the information in Tables 3.3 and 3.5. Values of 2.0×10^4 $\mu g\text{-persons}/m^3$ for Ni and 940 $\mu g\text{-persons}/m^3$ for Pb are obtained. These values are rough approximations only, because, among other things, they include only those cycle stages for which Pb or Ni emission data are available. Also, these values would be much smaller under a no-growth scenario.

References for Section 3.

1. R.K. Sharma, et al, "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," Argonne National Laboratory, ANL/ES-90, Argonne, IL, May 1980.

2. Personal Communication from M. Bernard, EES Division, Argonne National Laboratory, Argonne, IL, 1980.

3. R. Loutfy, E. Hayes, D. Graczyk, R. Varma, F. Williams, and N.P. Yao, "Stibine/Arsine Monitoring During Electric Vehicle Operation, Preliminary Results at ANL, Interim Report No. 1," Chemical Engineering Division, Argonne National Laboratory, Argonne, IL, June 1980.

4. A.C. Simon, "Stibine Generation in the Lead-Acid Battery," in "Stibine Formation and Detection in Lead-Acid Batteries," W.C. Spindler (ed.), Electric Power Research Institute, Report No. EPRI EM-448-SR, Palo Alto, CA, May 1977.

5. R. Varma and N.P. Yao, "Stibine and Arsine Generation for a Lead Acid Cell During Charging Made Under a Utility Load-Leveling Duty Cycle," Argonne National Laboratory, ANL/OEPM-77-5, Argonne, IL, March 1978.

6. Personal Communication with Marvin Wesely of the RER Division, Argonne National Laboratory, Argonne, IL, 1980.

7. J.G. Cleland and G.L. Kingsbury, "Multimedia Environmental Goals for Environmental Assessment," U.S. Environmental Protection Agency, Vols. I, II. Report Nos. EPA-600-7-77-136a/b, Research Triangle Park, NC, 1977.

8. A.J. Dvorak, et al., "Impacts of Coal-Fired Power Plants on Fish, Wildlife and Their Habitats," U.S. Department of the Interior, Fish and Wildlife Service, FWS/OBS-78/29, Washington, DC, 1978.

9. G.C. Holzworth, "Meteorological Potential for Urban Air Pollution in the Contiguous United States," Paper (NE-20c) presented at the Second International Clean Air Congress, Washington, DC, December 6-11, 1970.

10. Versar, Inc., "Assessment of Industrial Hazardous Waste Practices in the Storage and Primary Battery Industries," U.S. Department of Commerce, Report No. PB-241-204, prepared for U.S. Environmental Protection Agency, January 1975.

4. ECOSYSTEM EFFECTS

Quantification of environmental impacts from the lead/acid, nickel/zinc, and nickel/iron battery cycles is limited by a paucity of information relating environmental responses to emission levels and an inherent imprecision of our knowledge about emission levels.[1] Future control technologies and regulations, future battery technologies, and actual levels of emissions are uncertain. Because of these problems, assessments presented in this section cannot give a precise prediction of expected impacts. The estimate of impacts serves primarily to identify those aspects of the battery cycles likely to lead to environmental problems.

4.1 ESTIMATED PERMISSIBLE CONCENTRATIONS

Estimated permissible concentrations (EPCs) of battery-related emission constituents have been used as a guide in evaluating environmental impacts of battery cycles. Estimated permissible ambient concentrations (EPCs) of emission constituents from battery cycles are shown in Table 4.1. The data are derived principally from a review by Cleland and Kingsbury for the EPA.[2] The EPC values are derived from EPA secondary standards, recommended criteria, or minimum levels known to be toxic to vegetation, and are lower than threshold values required to produce toxic effects. Because the effects of long-term exposures of organisms in nonlaboratory situations are poorly understood, safety factors have been applied to toxicity data in the derivation of the EPC values.

EPCs were chosen for several reasons:

- It is beyond the "state-of-the-art" to develop dose-response relationships for environmental assessment in the total environment.

Table 4.1. Estimated Permissible Ambient Concentrations (EPC) of Emissions from the Battery Cycles for Protection of the Environment[a]

Emissions	Air, $\mu g/m^3$	Water, $\mu g/L$	Land, $\mu g/g$
Gaseous			
Cl_2	150[b]	2[c]	-
CO_2	10,000	30	-
Propylene	17,000	> 50,000	> 1,000
SO_2	160[d]	-	-
Particulate			
Total Partic.	150[e]	-	-
As	0.005[f]	10	0.02
Cd	0.12[f]	0.4	0.0004
Co	0.12[f]	50	0.1
Cr	0.12[f]	50	0.1
Cu	0.5[f]	10	0.2
Fe	16,000[g]	1,000[c]	2[c]
Hg	1	50	0.1
KOH	5[f]	75[f]	0.15[f]
Li	0.05[f]	75	0.15
Mg	14[f]	43,000	87
Mn	12[f]	20	0.04
Ni	0.24[f]	2	0.004
Pb	0.36[f]	10	0.02
Sb	1.2[f]	40	0.08
Zn	9.5[f]	20	0.04

[a]Primary data source: J.G. Cleland and G.L. Kingsbury, "Multimedia Environmental Goal for Environmental Assessment, Vol. II. MEG Charts and Background Information," U.S. Environmental Protection Agency, Washington, DC, EPA 600/7-77-136b, 1977.

[b]Based on data from "Chlorine and Hydrogen Chloride," National Academy of Sciences, Washington, DC, 1976.

[c]From "Quality Criteria for Water," EPA 440/9-76-023, U.S. Environmental Protection Agency, Washington, DC, 1976.

[d]U.S. Environmental Protection Agency 3-h secondary standard normalized to 24-h.

[e]U.S. Environmental Protection Agency 24-h secondary standard.

[f]Value not available for ecological effects; it is based on health effects.

[g]Based on data from "Subcommittee on Iron, Committee on Medical and Biological Effects of Environmental Pollutants," National Research Council, University Park Press, Baltimore, Maryland, 1979.

- Minimum toxic levels are highly species-specific and highly dependent on abiotic conditions and therefore extrapolation to different species under different abiotic conditions can be very misleading.

- Toxicity data are for acute toxicity, whereas chronic exposures are the most likely problem areas in context of this program. Therefore, basing analyses on acute exposures could underestimate the problem.

- EPCs attempt to translate from acute exposure to chronic exposure.

- EPCs are lower than minimum acute toxicity levels, usually by at least an order of magnitude.

- EPCs, as used for this program, are "indicator levels" as opposed to "permissible levels." As a result, if a given pollutant concentration exceeds the stated EPC, an adverse impact will not necessarily occur; rather, a potential for deleterious effect from this pollutant is indicated, which requires further scrutiny.

Concentrations of potentially hazardous constituents in the ambient atmosphere, soil, and runoff are assumed to be zero for simplification of analyses, even though potentially toxic materials probably are already present. Interactions (antagonistic, additive, or synergistic) between emission constituents have not been considered, because few data exist on these complex relationships. These interactions could result in toxic effects even though EPCs for individual constituents are not exceeded.

Assuming that wastewaters from battery-related cycles are discharged into streams, the following relationship is used to predict receiving-stream flow rates required to achieve acceptable stream EPC values for potentially toxic discharge constituents with no sedimentation after complete mixing:

$$EPC = \frac{C_r D_r + C_e D_e}{D_r + D_e} ,$$

where EPC is the estimated permissible concentration of a given parameter in the receiving stream after complete mixing; C_r is the ambient receiving-stream concentration of a given parameter before effluent addition, assumed to be zero for simplification of analyses; C_e is the effluent concentration of a given parameter; D_r is the receiving stream flow rate; and D_e is the effluent flow rate. Ambient receiving stream concentrations of potentially toxic constiturents probably are present. The presence of potentially toxic materials in receiving streams would necessitate additional dilution to achieve acceptable stream EPC values. Because complex interactions can occur between discharge constituents and receiving stream biota, an additive relationship was assumed; therefore, receiving-stream flow rates for each constituent were summed. The degree of environmental degradation from battery-related industries will depend on the quantity and quality of discharge and on receiving-stream flow rates.

4.2 EXPOSURE TO EMISSIONS

The estimated quantity of emissions for each phase of three battery cycles (lead/acid, nickel/zinc, and nickel/iron) were projected for the year 2000. The amount of ecosystem exposure to each of the emission products, up to 50 km from the source, is presented in relation to the EPCs (Table 4.1). A detailed dispersion analysis is presented in Sharma et al.[1]

4.2.1 Lead/Acid Battery Cycle

Emissions from model lead mining and milling, primary lead smelting, lead/acid battery manufacturing, and secondary lead smelting industries appear to present potential for causing environmental degradation (Table 4.2). Lead appears to be the most potentially hazardous constituent. Total atmospheric emissions of several constituents from battery-related complexes and resulting pollutant concentrations in air, soil, and runoff are presented in Figures 3.1 through 3.4.

The model lead mine-mill complex wastewater discharges appear to be the only battery-related industry discharges which have the potential to cause serious environmental degradation (Table 4.3). No discharges are expected from secondary lead smelting operations.

Table 4.2. Maximum Distances (km) from Model Lead/Acid Battery-Related Industries at which Estimated Permissible Concentrations of Identified Emission Constituents Are Exceeded

Emission Constituent	Lead Mining and Milling			Primary Lead Smelting			Lead/Acid Battery Manufacturing			Secondary Lead Smelting		
	Air	Land	Water	Air	Land	Water	Air	Land	Water	Air	Land	Water
CO	NE[a]	-	20-30	-	-	-	-	-	-	-	-	-
SO_2	-	-	-	-	-	-	-	-	-	NE	-	-
As	-	-	-	0-1	8-10	0-1	NE	NE	NE	NE	1-2	0-1
Cd	2	30-50	30-50	-	-	-	-	-	-	-	-	-
Co	2	10-15	2-4	-	-	-	-	-	-	-	-	-
Cu	4	30-50	30-50	-	-	-	-	-	-	-	-	-
Fe	NE	30-50	8-10	-	-	-	-	-	-	-	-	-
Mn	NE	30-50	20-30	-	-	-	-	-	-	-	-	-
Ni	1	30-50	20-30	-	-	-	-	-	-	-	-	-
Pb	50	30-50	30-50	30	30-50	30-50	2	30-50	15-20	10	30-50	30-50
Sb	-	-	-	-	-	-	NE	0-1	0-1	NE	10-15	1-2
Zn	4	30-50	30-50	-	-	-	-	-	-	-	-	-

[a]Non-exceedence at 1 km or 0-1 km.

Table 4.3. Receiving-Water Flow Rates Required for Dilution of Model Lead/Acid Battery-Related Industry Discharges to Achieve Acceptable Ambient EPCs for Protection of the Environment[a]

Model Industry	Model Industry Capacity, Equivalent MWh	Flow Rate, m^3/s
Lead mine-mill complex	12,295	86
Primary lead complex	18,750	4
Lead-acid battery manufacturing plant	4,000	
Dry process and $Ca(OH)_2$ treatment		2
Dry process and NaOH treatment		1
Wet process and $Ca(OH)_2$ treatment		1
Wet process and NaOH treatment		1
Lead-acid battery-breaking complex	5,769	0.1

[a]Derived from Sharma et al., "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," Argonne National Laboratory, ANL/ES-90, 1980.

Model polypropylene manufacturing industry atmospheric emissions, and resulting soil and runoff constituent concentrations are not expected to exceed EPC values. Polypropylene industry wastewaters have been poorly characterized and quantified; it is known, however, that wastewaters contain suspended solids, vanadium, titanium, aluminum, solvents, and inhibitors, in unknown quantities.

The major emissions from projected EV battery use will result from arsine and stibine production during battery charging. These gases are extremely toxic and decompose rapidly to oxide dusts; both gases and oxides could collect in closed garages and present human health problems, but are not expected to pose major environmental problems. Another potential hazard is the production of explosive hydrogen and oxygen gases during charging, but these are not expected to pose major environmental problems. Ozone is produced in small quantities during EV operation, but is unlikely to cause environmental problems. Indirect effects may be attributed to facilities that generate electricity for recharging batteries.

Information concerning atmospheric discharges from battery breaking is unavailable, but the potential quantities to be emitted are considered small. Little dilution of wastewater discharges (Table 4.3) is required to achieve acceptable EPC values.

4.2.2 Nickel/Zinc Battery Cycle

Emissions from model zinc mining and milling, zinc smelting, nickel smelting, and nickel/zinc battery manufacturing industries appear to present potential for causing environmental degradation (Table 4.4). Cadmium, cobalt, nickel, lead, sulfur, and zinc appear to be the most potentially hazardous constituents.

Total atmospheric emissions from battery-related complexes and resulting soil and runoff constituent concentrations are presented in Figures 3.5 through 3.8. Several model industry wastewater discharges appear to have the potential to cause serious environmental degradation (Table 4.5). The model nickel smelter is estimated to require a receiving stream flow rate of 400 m^3/s to achieve acceptable EPC values. This receiving flow rate, however, is based on data from a Canadian operation. Dilution requirements based on U.S. EPA effluents would be similar to requirements for the zinc industry. Additionally, future regulations for the nickel/zinc battery manufacturing industry probably would restrict effluent discharges.

Emission values for nickel mining and milling are not as well defined as for zinc production. The nature of the nickel tailings is not quantified, but it is expected that other elements in addition to nickel, such as copper, lead, and cadmium, will be the major potential pollutants.

Projected ground-level atmospheric concentrations of chlorine and carbon monoxide near a model caustic potash production plant are several orders of magnitude below the EPCs for environmental protection. The major toxic element in wastewater effluent is lead. Assuming that emissions meet EPA water effluent standards, a relatively low flow is required to achieve acceptable EPC values in receiving waters (Table 4.5).

Table 4.4. Maximum Distances (km) from Model Ni/Zn Battery-Related Industries at which Estimated Permissible Concentrations of Identified Emission Constituents are Exceeded

Emission Constituent	Zinc Mining and Milling			Zinc Smelting			Nickel Smelting			Nickel/Zinc Battery Manufacturing		
	Air	Land	Water	Air	Land	Water	Air	Land	Water	Air	Land	Water
SO_2	-	-	-	6	-	-	50	-	-	-	-	-
As	-	-	-	NE[a]	6-8	0-1	-	-	-	-	-	-
Cd	NE	15-20	1-2	NE	30-50	30-50	-	-	-	-	-	-
Co	NE	0-1	NE	-	-	-	10	30-50	15-20	1	8-10	1-2
Mn	NE	15-20	2-4	-	-	-	-	-	-	-	-	-
Ni	NE	2-4	0-1	-	-	-	50	30-50	30-50	6	30-50	30-50
Pb	1	30-50	10-15	NE	15-20	2-4	10	30-50	30-50	-	-	-
Sb	-	-	-	NE	1-2	NE	-	-	-	-	-	-
Zn	NE	20-30	4-6	NE	30-50	30-50	-	-	-	NE	30-50	20-30

[a]Non-exceedence at 1 km or 0-1 km.

Table 4.5. Receiving-Water Flow Rates Required for Dilution of Model Ni/Zn Battery-Related Industry Discharges to Achieve Acceptable Ambient EPCs for Protection of the Environment[a]

Model Industry	Model Industry Capacity, equivalent MWh	Flow Rate, m^3/s
Zinc mine-mill complex	43,000	43
Canadian nickel operation	100,000	400
Primary zinc complex	125,000	75
Caustic potash operation	100,000	0.004
Nickel-zinc battery manufacturing plant	4,000	3,200

[a]Derived from Sharma et al., "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," ANL/ES-90, 1980.

Model polypropylene manufacturing industry atmospheric emissions and resulting soil and runoff constituent concentrations are not expected to exceed EPC values. Polypropylene industry wastewaters have been poorly characterized and quantified; it is known, however, that wastewaters contain suspended solids, vanadium, titanium, aluminum, solvents, and inhibitors in unknown quantities.

Direct environmental effects from battery use are expected to be immeasurable because of the essentially emission-free operation of the battery system. Indirect effects may be attributed to facilities generating electricity for recharging batteries.

Emissions from the materials-recovery process of Ni/Zn batteries have not been quantified because of uncertainties surrounding technologies that will be used. Predominant air emissions will be fugitive dusts containing zinc, nickel, and other metals. These probably would not be spread over a large area because the batteries must be kept wet to reduce the chances of zinc combustion. Major environmental problems could be associated with liquid and solid wastes associated with recovery. Ni, Zn, Co, and Cd are expected to be the major factors posing an environmental threat.

4.2.3 Nickel/Iron Battery Cycle

Emissions from model nickel smelting, iron and steel production, and nickel/iron battery manufacturing industries appear to present potential for causing environmental degradation (Table 4.6). Emissions from the iron and steel industry are unlikely to be measurable above accumulation due to current steel production. Nickel, lead, and cobalt appear to be the most potentially hazardous constituents. The nickel and battery manufacturing industry wastewater discharges appear to have the potential to cause serious environmental degradation unless properly controlled (Table 4.7).

Maximum expected concentrations of particulates from the model LiOH facility are several orders of magnitude below the EPC for protection of the environment. However, particulate emission composition has not been quantified. Impacts of accumulation of particulates in soils and water cannot be assessed further without knowledge of particulate composition, nor can the impacts of solid waste be quantified. No wastewater discharges are expected.

Projected ground-level atmospheric concentrations of chlorine and carbon monoxide near a model caustic potash production plant are several orders of magnitude below the EPCs for environmental protection. The major toxic element in wastewater effluent is lead. Assuming that emissions meet EPA wastewater effluent standards, a relatively low flow is required to achieve acceptable EPC values in receiving waters (Table 4.7).

Model polypropylene industry atmospheric emissions and resulting soil and runoff constituent concentrations are not expected to exceed EPC values. Polypropylene industry wastewaters have been poorly characterized and quantified; it is known, however, that wastewaters contain suspended solids, vanadium, titanium, aluminum, solvents, and inhibitors in unknown quantities.

As with the other battery systems, direct effects from use of the Ni/Fe battery are expected to be negligible. Major effects will be indirect from generation of electricity for recharging.

Table 4.6. Maximum Distances (km) from Model Ni/Fe Battery-Related Industries at which Estimated Permissible Concentrations of Identified Emission Constituents Are Exceeded

Emission Constituent	Nickel Smelting			Iron and Steel Production			Nickel/Iron Battery Manufacturing		
	Air	Land	Water	Air	Land	Water	Air	Land	Water
SO_2	50	-	-	-	-	-	-	-	-
Cr	-	-	-	NE[a]	2-4	NE	-	-	-
Co	10	30-50	15-20	-	-	-	-	-	-
Cu	-	-	-	NE	2-4	2-4	-	-	-
Fe	-	-	-	-	-	-	NE	30-50	1-2
Mn	-	-	-	NE	30-50	2-4	-	-	-
Ni	50	30-50	30-50	NE	10-15	1-2	-	-	-
Pb	10	30-50	30-50	NE	15-20	15-20	-	-	-
Sb	-	-	-	-	-	-	-	-	-
Zn	-	-	-	NE	30-50	20-30	-	-	-

[a]Non-exceedence at 1 km or 0-1 km.

Table 4.7. Receiving-Water Flow Rates Required for Dilution of Model Ni/Fe Battery-Related Industry Discharges to Achieve Acceptable Ambient EPCs for Protection of the Environment[a]

Model Industry	Model Industry Capacity, equivalent MWh	Flow Rate, m^3/s
Canadian nickel operation	100,000	400
Lithium mine-mill complex	40,000	0
Iron-steel factory	200,000	0.47
Caustic potash operation	100,000	0.004
Nickel-iron manufacturing plant	4,000	40

[a]Derived from Sharma et al., "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," ANL/ES-90, 1980.

As with the Ni/Zn battery system, it is impossible at this time to present a specific assessment of emissions from Ni/Fe battery recovery. In general, the impacts from these emissions should be similar to those associated with the other battery systems. Iron would replace zinc as a major component of the emissions, but iron is generally considered less toxic. Lithium would also be a major component of emissions. Effects from electrolyte misting and disposal are expected to be of less magnitude than with the Ni/Zn system. It is anticipated that most of the electrolyte will be discarded prior to transport to the recovery operation and impacts would be mitigated by dispersing the recycling operation over a greater number of sites.

4.3 PROJECTED IMPACTS

Of the identified constituents emitted during the battery cycles, only a few are believed to represent a potential hazard to the environment according to projections in this report (Sb, As, Cd, Pb, Ni, S, Zn). The biogeochemistry of these elements is presented in Sharma et al.[1]

Soils and sediments serve as sinks for many constituents released from battery-related industries. Although increases of constituents in soils and sediments may be small, these concentrations will increase with time, and the likelihood of adverse environmental impacts will increase with continued deposition. This analysis emphasized the annual increment of increase in metal concentration. Accumulation will occur over the lifetime of the facility. The magnitude of the impacts will vary from site to site and depends on prior rates of metal deposition, soil and water-body properties, background concentrations of elements in the environment, and leaching and sedimentation rates. Where annual incremental additions to the soils occur, cumulative effects of metals can be expected, particularly where leaching and mobilization rates are low. The two major direct effects of SO_2 pollution of soils are a decrease in pH and an increase in sulfate.[1]

Elements emitted to the environment from the battery cycles will be cycled among biota and their physical environments to some degree. However, this cycling will be highly site- and species-specific. High concentrations of these elements can retard germination and inhibit nutrient recycling, resulting in disruption and possible elimination of ecosystem structure and function. Bioconcentration of these elements above ambient levels is likely in aquatic ecosystems but not in terrestrial ecosystems, although in terrestrial ecosystems higher-than-normal tissue concentrations could occur in areas contaminated with these elements. Bioconcentration of potentially toxic elements can result in acute and chronic toxicity to exposed biota. Biomagnification of elements along the food chain leading to man is not likely to be important for the major constituents of the emissions from the battery cycle.[1]

Solid wastes from battery-related industries could contain potentially hazardous constituents, and adverse impacts could result from the leaching of these wastes into soils, groundwater, and surface water. Leaching of potentially toxic constituents from settling ponds and other treatment ponds into ground and surface waters could have adverse impacts. Leaching rates are dependent on a host of environmental variables; however, proper management of waste materials in accordance with the Resource Conservation and Recovery Act of 1976 (P.L. 94-580) should mitigate adverse impacts.

Groundwater supplies could be adversely affected, both quantitatively and qualitatively, by intersecting aquifers during mining operations. Physical surface disturbances resulting in habitat destruction also will occur as battery-related industries are established, especially mines and mills, and as solid wastes (mining overburden, mill tailings, sludge from treatment processes, and slag) are disposed. However, land reclamation should mitigate effects associated with surface disturbances. Long-term land allocation will be required for many battery-related industries.

Direct environmental effects from battery use are expected to be immeasurable because of the essentially emission-free operation of the battery system. Indirect effects may be attributed to facilities generating electricity for recharging batteries.

References for Section 4

1. R.K. Sharma et al., "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Battery Cycles," Argonne National Laboratory, ANL/ES-90, Argonne, IL, 1980.

2. J.G. Cleland and G.L. Kingsburg, "Multimedia Environmental Goal for Environmental Assessment, Vol. II. MEG Charts and Background Information," U.S. Environmental Protection Agency, EPA 600/7-77-1366, 1977.

5. HUMAN HEALTH RISKS

In previous sections, the compositions of the three near-term EV batteries have been described, in addition to the industrial support system required for their production, the emissions associated with the industries, and the dispersion of these emissions into the environment around the major industrial facilities. In this section the latter information is related to an estimate of risk to human health via several approaches designed to address the health risks to the various segments of the affected population and provide a range of reasonable estimates.

The first approach (Sec. 5.1) utilizes human dose-response functions developed from analysis of the toxicological and epidemiological literature concerning exposure to the toxic elements of interest. A dose-response function has been developed for lead because lead is in the air around mining and milling complexes and primary and secondary smelting and refining operations and is therefore breathed by populations living and working in the vicinity of these operations. In addition, 50% to 100% increases in the industries required for lead/acid battery manufacture are anticipated (Table 3.2). Cadmium and arsenic are toxic contaminants present in the emissions from mining, milling, and smelting of lead and zinc ores. Arsenic also is a component of the lead electrode of the lead/acid EV battery. Dose-response functions for these two elements have also been developed. A fourth major element is nickel; a causal relationship has been indicated between human exposure to nickel compounds and development of respiratory tumors. The analysis for nickel is underway and is not presented in the current document.

The dose-response approach uses the environmental dispersion information presented in Section 3 to predict incidences of adverse health effects among persons exposed to emissions at various distances from the battery-related industrial operations.

The second approach examines effects among the occupationally exposed through (1) application of the dose-response models to possible workplace exposures, and (2) analysis of data on rates of occupational injury and illness. The final sections describe the effects of acute and chronic exposure to stibine, arsine, and antimony trioxide and relate these effects to potential exposures during charging of lead/acid EV batteries.

5.1 ENVIRONMENTAL EXPOSURES

5.1.1 The Risk-Analysis Approach

The health risks from various energy technologies can be divided between those primarily of a safety nature, such as individual accident hazards, and those associated with exposure to low levels of toxic agents. Both situations involve exposure of a population to a physical hazard. Safety risks, however, tend to be limited to a smaller population, either in the workplace or under unique circumstances that place individuals in the general public in proximity to activities associated with continuous operation of the industry. Risks from exposure to toxic agents, although they tend to be more subtle and much more difficult to quantify than safety risks, can have grave implications to large segments of the population. Our success in estimating the societal costs of future technical advances and industrial expansions, such as those proposed for future energy production and use, will be limited by our ability to quantify the health risks of low-level exposure to toxic agents.

Analysis of the level of risk to health posed by a developing technology can be accomplished only within limited confidence bounds. Such analyses depend on the associations between specific agents from the proposed technology and unique health states. All too often the causal relationship of such an association is only superficially understood. Further, exposure levels are at best measured to an order of magnitude and even at that are subject to the whims of meteorology and topography. Nevertheless, emerging technologies such as those of the various energy options demand that we attempt at least preliminary estimates of their impacts on human health. In this situation we are not allowed the luxury of waiting for definitive findings of the causal relationship nor can we presume such an analysis to be impossible. The

question we face is, therefore, not whether it is possible to assess the potential level of hazard but rather how well such an estimate can be made given the present state of the informational basis upon which such estimates are generated.

Under these circumstances predictions of impact which incorporate the rules of probability provide a numerical description for the expression of the confidence in predicted risk levels. This approach assumes the use of informed judgment to extrapolate from the probabilistic estimates of risk to the actual level of hazard experienced by a single individual. In this section we utilize what is known about the various techniques of risk estimation to provide a quantitative judgment of the level of hazard to populations environmentally and occupationally exposed to emissions from energy storage technologies. For the purposes of this analysis it is of value to consider the implications of the most conservative yet plausible models that supply quantitative expressions of health risk. A supporting document detailing the assumptions of the approach is now in preparation.[1]

In proposing a technique of risk estimation, the nature of the induced response must be considered as an additional factor in the association. For the most part an agent that induces a lethal response is much more conducive to quantification than one which results in a sublethal chronic disease response, since the latter has a less clearly defined endpoint. It is also very likely that a sublethal, rather than a lethal, response may result in the greater overall societal impact by incurring increased medical care on the population.

The heavy metals are conservative environmental contaminants. That is, unlike gaseous pollutants with well-defined half-lives by which they decay to less toxic compounds and by which an environmental concentration would eventually decay to zero, the elemental agents such as the heavy metals can be expected to have their environmental concentrations increase with time or at least maintain a steady-state concentration. The biological significance of toxic heavy metals also is important in that they serve no known purpose in the various physiological systems. These systems are often without an efficient process for clearance or detoxification, and as a result the metals tend to build in concentration within specific sites in the body.

In the present document, focus is placed on three metals involved in the near-term electric vehicle storage battery cycles: lead, arsenic, and cadmium. The respective toxic endpoints are central nervous system dysfunction, respiratory cancer, and proteinuria. Exposure estimates are derived from airborne concentrations of metals from point source emissions for specific industrial operations in the battery cycles described in Section 3. In what is today a standard approach to the problem, emphasis has been placed on the local area effect; exposure and subsequent response are determined at distances of 5, 15, and 30 km from a typical battery-industry site. Long-distance dispersal has also been evaluated.

5.1.2 Lead

Lead toxicity is commonly believed to manifest itself first in the bone marrow, the critical organ of blood formation.[2] Lead inhibits nearly all the enzymatic steps that lead to heme synthesis. It reacts with the reticutocyte membrane and intracellularly with mitochondria, whose presence in the immature red cell is required to provide the necessary enzymatic machinery for synthesis of heme.[3]

Response to lead absorption is graded and related to the magnitude of exposure, where effects represent a continuous spectrum ranging from subtle changes within homeostatic bounds to severe injury.[4] As a result, the onset of lead toxicity, even when acute, is not a sharply defined event; rather, it involves a continuum of change from normal to ill health.[5] However, the deleterious effect of lead on heme biosynthesis provides a biochemical basis for the evaluation of man's response to lead exposure even before clinical manifestations of lead poisoning are present.[6,7] The use of minor hematological changes as measures of potential health risk is in accordance with the World Health Organization position that, whenever possible, health risk estimation should be based on parameters that serve as a warning signal for impending risks.[8] In this regard measurement of metabolites or biochemical changes should be quantitatively related to an adverse health effect.

The approach taken in the present analysis utilizes the association of continuous blood-lead (Pb-B) to erythrocyte protoporphyrin (EP) concentrations

in circulating red blood cells. EP changes occur slowly in the bone marrow from which mature red cells are released into the peripheral circulation (circulating blood). Thus, there is a time lag between exposure and increase of peripheral blood EP. Because EP remains in the erythrocyte (red blood cell) throughout its lifetime (about 120 days), its assay is an integrated measure of chronic effect. Recent studies have confirmed that the change in EP concentration represents a long-term and relatively stable effect, and that these changes are slow and predictable.[9] Further, it is now acknowledged that when EP suggests a different clinical category from Pb-B, the Pb-B level is likely to change in the direction suggested by EP.[10]

Thus, in this analysis a predicted EP response to a specific airborne lead concentration is used to estimate the level of risk associated with a prolonged lead exposure. Using a Pb-B value greater than 60 mg/dl as an indicator of substantial health risk for central nervous system dysfunction, it is possible to derive an estimate of likelihood that an individual is at or above this hazard level. The estimate is based on a generalized dose dependency of EP to Pb-B of the form: $EP = ae^{b(Pb\text{-}B)}$. The model incorporates assumptions on the general form of the EP and Pb-B distributions in a population exposed to airborne lead and in a control population. The relationship between EP and Pb-B is used to derive an estimate of mean level of risk. The relationship also is the basis for generating statistical confidence intervals about the mean estimated response level. Details are given in Appendix A.

Dispersion plots of airborne lead concentrations from point source emissions of specific industrial operations in the manufacture of lead/acid storage batteries have been used to derive exposure estimates at distances of 5, 15, and 30 km from the emission site. In the analysis three types of industrial operations were assessed; a mining and ore milling operation, a primary lead smelter, and a secondary lead smelter associated with a battery manufacturing plant. The overall level of health risk for the general public, as defined above, is given in Table 5.1. The individual risk estimates can be interpreted in terms of population risk by multiplying by an appropriate population density factor. These calculated values represent the level of risk associated with a continuous annual exposure to airborne lead particulates. The extremely high value associated with the 5-km location from the

Table 5.1. Calculated Health Risks Associated with Continuous Exposure to Airborne Lead Concentrations

Industrial Operation	Distance from Point Source (km)	Lead Conc.[a] ($\mu g/m^3$)	Standard Error		Individual's Probability of a Pb-B>60 $\mu g/dl$	Upper 95% Confidence Limit of Estimate[b]
			S_{EP}	ln S_x		
Ore mining and milling	5	12.5	30.9[c]	2.73[d]	0.0032	0.23
	15	2.8	26.4	2.90	0.0019	0.07
	30	0.8	23.7	2.96	0.0015	0.03
Primary smelter	5	4.2	27.4	2.86	0.0021	0.09
	15	1.3	24.7	2.92	0.0018	0.04
	30	0.36	22.4	2.99	0.0014	0.02
Secondary smelter and battery mfg. plant	5	1.36	24.7	2.94	0.0016	0.04
	15	0.29	22.0	3.00	0.0013	0.02
	30	0.10	20.6	3.01	0.0013	0.01
Reference	e/	0.353			0.0012	0.01

[a] Lead concentrations are derived from Figure 3.2. A reference background concentration (0.353 $\mu g/m^3$) is added prior to calculation of the health risk.

[b] Lower confidence limit is assumed to be below that of a background level of risk.

[c] Standard error for erythrocyte protoporphyrin concentration.

[d] Geometric standard error for blood lead concentration.

[e] Background risk levels are approached at approximately 50 km from the mine, mill site. For the other operations, a shorter distance would be required to reach background levels. The long distance transport of Pb in air would therefore be expected to be of negligible incremental effect over background environmental exposure effects.

ore mining site would imply a substantial community health risk. Such an exposure situation would not be expected to occur under the present air quality standard without some form of regulatory intervention. In this regard, the calculated level of risk may be somewhat unrealistic. For the other exposure situations, however, the calculated risk level is assumed to represent a reasonable estimate of a worst-case exposure scenario. It should be noted that long-distance air transport of lead from these operations is anticipated to have negligible incremental effects over background.

5.1.3 Arsenic

Arsenic may cause both acute and chronic intoxication. Subacute and chronic poisoning usually stems from exposure to contaminated air and drinking water. Acute arsenic poisoning is rare and is seen only in connection with homicidal or accidental poisoning by ingestion. The acute lethal dose is estimated to be in the range of 70-180 mg. Chronic arsenic poisoning in workers with long-term inhalation exposure is characterized by skin and mucus membrane lesions. Vascular insufficiency leading to gangrene in the extremities is a manifestation of systemic disease in endemic arsenic poisoning from drinking water.

Arsenic has toxicological manifestations throughout the central and peripheral nervous system of humans and is a cardiotoxin that mimics ischemic heart disease. It is also toxic to the kidneys. Arsenic has long been suspected of carcinogenic potential and only recently has its role as a human carcinogen been clarified. Arsenic trioxide is now increasingly accepted as an occupational carcinogen of the lung, skin, and possibly the liver. The evidence of its carcinogenic potential is unusual in that it is entirely dependent upon human epidemiological and clinical findings, which are unsupported by any experimental evidence in animals. The fact that no animal model for arsenical cancer has been established thus far has been an important factor in the delay of acceptance for the human cancer risk posed by arsenic exposure.

Three recent studies[11-13] support an exposure-dependent relationship between airborne arsenic trioxide and increased mortality from respiratory

cancer. In comparison to a nonoccupationally exposed population, smelter workers exposed to arsenic trioxide had an excess total mortality due mainly to malignant neoplasms of the respiratory system. One study[12] was able to eliminate smoking habits as the cause of the respiratory cancer death differential in arsenic workers. Another study[13] demonstrated that the mean latency period for the induction of the carcinogenic response is 30 years. These epidemiological studies have now established rather convincingly that human exposure to arsenic results in an excess risk of lung and lymphatic tissue cancers. Evidence from studies involving entirely different circumstances of exposure, such as workers in pesticide manufacturing plants, suggests that there is no essential cofactor for arsenic-induced respiratory cancer.

The NIOSH concensus is that the present state of knowledge is insufficient to demonstrate a safe level for carcinogenesis prevention. Prudence would therefore dictate that an absolute threshold cannot be established. Because of the seriousness of the disease, a minimum requirement for an exposure standard should be to significantly reduce the incidence of the induced response. Thus, the NIOSH position with regard to arsenic exposure is that the overwhelming evidence in man cannot be negated and arsenic is to be considered as a human carcinogen. Study results strongly suggest that exposure at or near 0.2 mg/m^3 of arsenic can result in increased incidence of lung cancer.[11] Based on the criteria outlined above, the existing federal standard for airborne arsenic has recently been updated and set at 0.05 mg/m^3 for workplace exposures.

The work of Pinto,[12] Lee,[11] and Ott[13] support the assumption of an exposure-dependent relationship between airborne arsenic trioxide and increased mortality from respiratory cancer. The association demonstrates a decidedly nonlinear function of the form:

$$y = e^{a} x^{b} ,$$

where y is the standardized ratio for mortality from respiratory cancer (ICD 160-164) and x is the annual mean exposure to arsenic trioxide measured in units of micrograms per cubic meter. The model developed for this health risk

analysis assumes an average latency period of approximately 30 years from initial exposure to the manifestation of the disease and a zero threshold for response.

In the case of an arsenic concentration derived from the dispersion calculations from a point source, estimates of the potential level of risk of respiratory cancer mortality in a local area can be estimated by the procedure outlined in Appendix A. For the present analysis, maximum ground level concentrations at 5, 15, and 30 km from a secondary lead smelter and a battery manufacturing plant, were incorporated into the model as the exposure parameters, and the estimated level of risk was calculated for a cohort exposed continuously to this level. The results are displayed in Table 5.2. This analysis suggests that if any risk from arsenic exposure exists at all within the 30-km radius, it is probably so small as to be negligible in comparison to the effect of general background levels of response.

Respiratory cancer response was selected as the indicator because of the possibility of a zero threshold. Other pathological responses are assumed to have "no response" levels. Therefore, an insignificant carcinogenic response implies, by definition in this analysis, that a significant arsenic-related health risk does not exist for the general population from the emissions of the point source used in this industrial definition. This is not to say that persons occupationally exposed will have an insignificant level of health risk.

Because a zero threshold model is assumed for the health response to arsenic exposure, a long-range dispersion scenario was analyzed for general population impact. In this situation a total storage battery industry emission is released from a continously operating point source, after which it is dispersed throughout the United States. For the sake of a conservative argument we assume the point source to be located on the west coast of the continental United States and allow the emissions to disperse to the eastern seaboard. Assuming a combined industrial emission, we estimate that some 1200 grams of arsenic are released into the atmosphere daily.

Table 5.2. Person-Years of Life Lost as a Result of Continuous Exposure to Airborne Concentrations of Arsenic (As_2O_3)[a]

Industrial Operation	Distance from Point Source, km	Arsenic Concentration, $\mu g/m^3$	Person-Years Lived[b]	Excess Person-Years Lost
Secondary smelter and battery mfg. plant	5	3.93×10^{-4}	99889.96	0.23
	15	9.6×10^{-5}	99890.13	0.06
	30	3.6×10^{-5}	99890.17	0.02
Reference[c]		6.6×10^{-2}	99890.19	

[a]Arsenic concentrations represented are those attributable to industrial operation or to background. For model calculations, total arsenic concentrations were obtained by adding background to incremental industrial operation values.

[b]Assumes a cohort of 10^4 persons exposed continuously for 30 years and subjected to an increased mortality rate to respiratory cancer for 10 years following cessation of exposure.

[c]Reference MR_r was chosen to be 7.14×10^{-4} deaths per person year from cancer of the lung, bronchus, and trachea for white male residents of the city of Baltimore, 1950-1974. From K. Mabuchi et al. "Lung Cancer among Pesticide Workers Exposed to Inorganic Arsenals," Arch. Environ. Health, 34(5):312-320, 1979.

The overall population response to the combined industrial release was estimated to be on the order of 0.003 cancer deaths per year in the total U.S. population. The impacts of various industrial component emissions were determined at: 0.002 cancer deaths from primary smelter emissions, 0.0008 cancer deaths from secondary smelter emissions, and less than 10^{-5} cancer deaths attributable to long-range transport of arsenic released during storage battery manufacture. Therefore, under the assumptions set forth in this analysis, the attributable health response of the total U.S. population, exposed to airborne arsenic from a storage battery industry, would be much less than one death from cancer of the lung, bronchus, and trachea per year.

5.1.4 Cadmium

The critical organs for cadmium toxicity are the kidney, liver, and lung.[14] The lung is generally only involved in occupational exposures via inhalation.[15] Because cadmium accumulates in the kidney and liver regardless of exposure route, these organs, particularly the kidney, are of concern in any cadmium exposure situation. About one-third of the total cadmium body burden is found in the kidneys,[14] primarily in the cortex,[16] where it competes with zinc for binding sites in enzymes and proteins.[17-21] At kidney cortex cadmium concentrations in the range of 100-300 μg/g wet weight,[14,22] the reabsorption capacity of the renal tubules is damaged and plasma proteins of low molecular weight begin to appear in the urine rather than being reabsorbed into the plasma as in normal persons. Animal studies support this damage hypothesis with evidence of organ tissue changes in the critical concentration range. Because it is not seen with other types of proteinurias, β_2-microglobulin (β_2-MG) in the urine is generally accepted as indicative of cadmium-induced renal tubular damage.[23-34] Levels of urinary β_2-MG of 400 μg/L or greater are indicative of clinical tubular proteinuria.[31] The damage is irreversible and may result in further dysfunction or additional renal damage.[14,22,32] Osteomalacia has been reported in cadmium workers[33] and Japanese with severe environmental exposures.[14]

The cadmium model developed for risk analysis is one in which daily exposure is related to a kidney concentration and subsequently to an average urinary protein level. A basic assumption is that cadmium exposure will result in an upward shift in mean concentrations of cadmium in the kidney for a population such that the individual risk is increased for clinically defined tubular proteinuria. In this analysis a conservative risk estimate was made using an epidemiological definition of tubular proteinuria based on the upper 95% confidence interval of a reference population.[34] A daily cadmium dose to the body can be calculated from ambient exposure levels through multiplication by the appropriate absorption and retention rates. Details of the model are presented in Appendix A.

In the analysis presented in Table 5.3, an initial estimate has been made of the excess risk of developing tubular proteinuria (conservative epidemiological definition) from ground-level cadmium concentrations at various distances from a lead mining and milling complex. Three distances are used: 5, 15, and 30 km from the complex. Risk is computed at the end of three exposure periods, 1, 10, and 30 years. Because cadmium accumulates over a period of years, it is anticipated that the risk would increase with the length of exposure. The actual level of excess risk is found by subtracting a normal probability value for a reference population from that derived for the exposed populations. Thus, the model assumes a background level of cadmium exposure present in the environment.

As was anticipated, the data shown for the local-area effect reflect an increased excess risk of tubular proteinuria with increasing cadmium levels associated both by proximity to the mining/milling complex and accumulation in the body over time. At the 5-km distance there is a considerable increase in risk after ten years (1-3 per 10,000). After a period of 30 years the excess risk is about 2 per 100, which would imply a significant level of risk. A considerable degree of risk, approximately 2 per 1000, is experienced by those persons at the farthest distance only after a period of 30 years. For those at the middle distance, risk also is most noteworthy at the 30-year point (3-4 per 1000), and moderate (2-4 per 10,000) at the ten-year point. It is estimated that the excess risk of tubular proteinuria would be 1×10^{-6} at the end of 30 years of exposure at a distance of 85 km from the mine/mill site. As was the case with arsenic, this is a nonsignificant effect.[13a]

5.2 OCCUPATIONAL HEALTH AND SAFETY

The occupational health and safety of workers in those industries required for large-scale production of near-term electric vehicle batteries is assessed by two different methods. The first method utilizes the toxicological models presented in Section 5.1 and Appendix A to predict responses to each of the three pollutants at exposure levels such as might be found in a

Table 5.3. Excess Risk of Developing Tubular Proteinuria from Ground-Level Cadmium at Various Distances from a Lead Mining and Milling Complex as Compared to a Reference Population

Distance from Complex, km	Ambient Cadmium Concentration, ng/m^3	Years of Exposure	Kidney Concentration, µg/g	β_2-Microglobulin, µg/L	Excess Risk of Developing Tubular Proteinuria[a]
5	42.5	1	1.458	14.522	0
		10	12.462	31.033	0.0013-0.0026
		30	27.192	85.732	0.0115-0.0207
15	14.1	1	1.382	14.445	0
		10	11.794	29.630	0.0002-0.0004
		30	25.674	77.206	0.0030-0.0037
30	9.7	1	1.370	14.433	0
		10	11.693	29.424	0-0.0004
		30	25.454	76.044	0-0.0015
Reference	7.5	1	1.364	14.428	
		10	11.643	29.322	
		30[b]	25.343	75.470	

[a]Range of values results from using standard error of two separate control populations.

[b]Excess risk of tubular proteinuria is 1×10^{-6} at a distance of 85 km from the mine/mill complex at the end of a 30-year exposure period.

typical battery-related industry work area. Assumptions made regarding the number of days and hours worked and exposure levels encountered outside the work area are described in Appendix A.4. The second method utilizes data from the U.S. Department of Labor, Bureau of Labor Statistics, on occupational injury and illness rates.[35] Occupational injuries account for 90% to 99% of the total cases reported; this method therefore addresses the issue of occupational safety and complements the first one, which assesses occupational health.

5.2.1 Occupational Health

Application of the arsenic dose-response function to possible workplace exposure levels results in a loss of 53.2 person-years from a total of 100,000 person-years. Lead exposure in a similar situation could result in a likelihood of 1 in 100 for having a blood-lead level at or above 60 μg/mL, the low cutoff point for central nervous system dysfunction. In the case of cadmium exposure, the excess risk of tubular proteinuria is about 0.325 after one year of exposure. It should be noted, however, that this is based on a very conservative definition of tubular proteinuria (> 290 μg β_2-MG/L urine).

5.2.2 Occupational Safety

A breakdown of the industries required for production of lead/acid batteries for electric vehicle use is presented in Table 5.4. The Standard Industrial Classification (SIC) code number for each industry is presented in addition to the level of output required for these industries by the year 2000 and per MWh to meet demands for electric vehicle battery production. Also listed are the number of new production units required to meet these demands. Injury and illness rates per 100 full-time workers for these industries are presented in Table 5.5. The incidence rates are presented along with comparison rates for persons employed in (1) all industries in the general SIC category related to the battery industry (GRI) and (2) the general private sector (GP). This comparison approach asks the question: Are there particular, identifiable risks associated with employment in industries required for electric vehicle battery production which are higher or lower than those which would be encountered by a work force of similar size employed in (1) a category

Table 5.4. Industrial Requirements for Production of Lead/Acid Batteries for Electric Vehicle Use

Industry	SIC Code	Output Required[a]		Number of New Production Units Required	
		By Year 2000, MT/day	Per MWh, kg/day/MWh	By Year 2000	Per MWh
Lead mining and milling	1031	1100 (Pb)	14.5	6.1[b]	8.1×10^{-5}
Primary lead smelting and refining	3332	1100 (Pb)	14.5	4.0[c]	5.3×10^{-5}
Secondary lead smelting and refining	3341	720 (Pb)	9.6	13[d]	1.7×10^{-4}
Lead/acid battery manufacture	3691	2800 (batteries)	28.5	23[e]	3.1×10^{-4}
Battery breaking	3341	720 (Pb)	9.6	13[f]	1.7×10^{-4}
Polypropylene industry	2821	180 (polypropylene)	2.5	0.78[g]	1.0×10^{-5}
Antimony smelting	3339	20 (Sb)	0.3	0.5[h]	6.7×10^{-6}
Sulfuric acid production	2819	1600 (H_2SO_4)	21.5	3[i]	4.0×10^{-5}

[a] Items to be produced are shown in parentheses in the first column. Electric vehicle fleet size by the year 2000 is taken as 3×10^6 vehicles (75,000 MWh) growing of 25% per year (ANL/ES-90, p. 8).

[b] The production rate of a large mine-mill complex is equivalent to 180 MT of lead produced/day (ANL/ES-90, p. 24).

[c] The primary smelter capacity is typically 100,000 MT Pb/year (ANL/ES-90, p. 28).

[d] The average secondary smelter size was taken to be 20,000 MT/year (ANL/ES-90, p. 61).

[e] A large battery plant produces 6500 standard auto batteries/day, each containing 11.8 kg of lead (ANL/ES-90, p. 45).

[f] The average secondary smelter size, which includes a battery breaker, was taken to be 20,000 MT/year (ANL/ES-90, p. 61).

[g] The average unit was taken to produce 85,000 MT/year (ANL/ES-90, p. 35).

[h] The one large antimony smelter in the U.S. produced 15,000 MT in 1974, or 40 MT/day (ANL/ES-90, p. 32).

[i] The average sulfuric acid plant produces 2×10^5 MT/year (ANL/ES-90, p. 33).

Table 5.5. Occupational Injury and Illness Rates for Industries Involved in Lead/Acid Battery Manufacture for Electric Vehicle Use

Industry	SIC Code	Incidence Rates[a] Sector	Total Cases	Lost Workday Cases	Lost Workdays	SIC Industry Description and Code Number[b]
Lead mining and milling	1031	SI[b]	6.5	4.4	69.2	Metal mining (10)
		GRI[b]	11.0	5.7	113.0	Mining
		GP[b]	9.1	3.3	56.1	Private sector
Primary lead smelting and refining	3332	SI	15.1	5.9	140.0	Primary nonferrous metals (333)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Secondary lead smelting and refining	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Lead/acid battery manufacture	3691	SI	23.5	10.0	176.0	Storage batteries (3691)
		GRI	11.0	3.8	66.6	Misc. electrical equipment and supplies (369)
		GP	9.1	3.3	56.1	Private sector
Battery breaking	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Polypropylene industry	2821	SI	8.8	2.8	43.5	Plastic materials and resins (2821)
		GRI	8.3	2.8	48.2	Chemical and allied products (28)
		GP	9.1	3.3	56.1	Private sector
Antimony smelting	3339	SI	20.0	10.1	194.3	Primary nonferrous metals, n.e.c. (3339)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Sulfuric acid production	2819	SI	8.1	2.6	53.0	Industrial inorganic chemicals, n.e.c. (2819)
		GRI	8.3	2.8	48.2	Chemicals and allied products (28)
		GP	9.1	3.3	56.1	Private sector

[a] Incidence rates represent the number of injuries and illnesses and lost workdays per 100 full-time workers in 1975, as calculated in U.S. Department of Labor, Bureau of Labor Statistics, 1978, Bulletin 1981.

[b] SI, GRI, and GP refer to specific industry, general related industry, and general population, respectively. The SIC industry description and code number used for these industry definitions are shown in the last column of the table.

of related industries requiring similar worker talents and experience and (2) the general working population?

Analysing the industries required for lead/acid battery manufacture (Table 5.5), we see that for those employed in lead mining and milling, polypropylene production, and sulfuric acid production, occupational injury and illness rates do not differ much from those in the general, related SIC industries, and from those in the private sector. In fact, employment in lead mining and milling has a lower injury and illness rate than does employment in the general mining category because of high injury and illness rates among anthracite miners and oil and gas extraction workers included in the general mining category.

For those employed in the primary smelting and refining of lead or antimony, a higher occupational injury and illness rate occurs compared to the average for the entire private sector; the incidence rate is not much higher, however, than the high rate in the general category of primary metal industries (SIC 33). If demand for lead/acid electric vehicle batteries pulls workers from the general primary metal industries to the primary smelting of lead or antimony, not much change in occupational injury and illness rates would be experienced. Workers in battery manufacture, battery breaking, and secondary smelting of lead, however, show a higher incidence rate than workers both in the general related industries and in the general private sector. Expansion of these industries to increase lead/acid electric vehicle battery production could result in a population of persons experiencing a rate of occupational injury and illness 1.5- to 2-fold higher than that of workers with similar skills employed elsewhere, and approximately 3-fold higher than the average for the entire private sector. A mean of 25% to 30% of the workers employed in the latter three battery-related industries could become injury or illness cases each year, with 10% of the workers requiring an average of 20 lost work days per injury or illness case per year (Table 5.5).

Industrial size requirements and incidence rate data for the nickel/zinc and nickel/iron systems, analagous to the information in Tables 5.4 and 5.5 for the lead/acid system, are included in Section A.5. of Appendix A. The findings of the present occupational safety study are summarized in Table 5.6

Table 5.6. Categorization of Industries Involved in Near-Term Electric Vehicle Battery Manufacture According to Incidence Rates for Occupational Injury and Illness

Battery System	Incidence Rates $\leq$ GP[a]	GP < Incidence Rates $\lesssim$ GRI[a]	Incidence Rates > GRI[a]
Lead-acid	Lead mining and milling	Primary lead smelting and refining	Secondary lead smelting and refining
	Polypropylene industry	Antimony smelting	Lead/acid battery manufacture
	Sulfuric acid production		Battery breaking
Nickel-zinc[b]	Zinc mining	Primary zinc smelting and refining	Secondary nickel refining
	KOH production	Nickel refining	Secondary cobalt refining
	Polypropylene production	Cobalt refining	
Nickel-iron[b]	Steel production	Nickel refining	Secondary nickel refining
	KOH production	Cobalt refining	
	LiOH production	Primary copper smelting and refining	
	Copper mining		
	Polypropylene production		

[a] GP and GRI refer to general population and general related industries, as in Table 5.5. Actual incidence rates for the categorized industries are given in Tables 5.5 and Appendix Tables A.3 and A.4.

[b] Battery manufacturing is not categorized for these battery systems.

by categorizing all industries required for near-term EV battery production according to whether they have incidence rates (1) less than or equal to those for persons employed in the general private section (2) greater than those for the private sector and approximately equal to those for persons employed in the general related SIC category, and (3) greater than those for persons employed in both the private sector and in the general related SIC category. The industries with occupational injury and illness rates greater than both comparison categories are those involved in secondary smelting and refining of metallic elements required for battery production and in lead/acid battery manufacture. Manufacture of nickel/zinc and nickel/iron battery systems is not included since incidence rates are not available for these industries.

Additional information must be obtained to convert the incidence rates presented here to total incidences of occupational injury and illness for a given work force size. An estimation must be made of the labor force requirement for the industry production levels designated in Table 5.4 and Appendix A.5. To do this, labor productivity estimates can be made similar to those described in the Photovoltaic Energy Technologies HEED. In the case of the lead/acid battery system, it is also possible that a typical labor force can be assigned to each typical new production unit required (Table 5.4).

5.3 HEALTH EFFECTS OF STIBINE AND ARSINE

Stibine and arsine are toxic gases generated during charging of lead-acid batteries containing antimony and arsenic (see Sec. 3). According to recent tests,[36] concentrations of stibine generated in the vehicle and in the garage during charging range from 2% to 17% of the current OSHA standard for occupational exposure to stibine (0.5 mg/m^3). Concentrations of arsine generated during charging range from less than 1% to 6% of the OSHA standard for arsine (0.2 mg/m^3). The 1975 NIOSH Criteria Document on inorganic arsenic has recommended a 100-fold reduction in the standard concentration for arsine, to 0.002 mg/m^3.[37] This reduction, if enacted, might significantly influence the feasibility of producing lead/acid batteries containing antimony and arsenic, since concentrations of arsine generated by present battery systems would exceed the new OSHA standard.

5.3.1 Metabolism

Animal experiments indicate that upon entry into the body, stibine and arsine concentrate mainly in the red blood cells and the liver.[38,39] Early elimination of antimony or arsenic following exposure to these gases is rapid, though incomplete. Guinea pigs eliminated approximately one-half of the deposited antimony within 45 minutes of exposure to 25 ppm stibine.[38] Mice eliminated 55% of the initially retained arsenic by 24 hours after exposure to 55 ppm arsine for 20 minutes.[39] Excretion of the arsenic remaining after early elimination was slow.

5.3.2 Acute Toxicity

Arsine is a highly poisonous, nonirritating gas with a mild garlic odor. Exposure to high concentrations results in sudden, extensive hemolysis (destruction of red blood cells).[40] Inhalation of 800 mg/m^3 (250 ppm) is reported as either instantly lethal,[41] or lethal after 30 minutes' exposure;[42] exposure to 80-160 mg/m^3 (25-50 ppm) for 30 minutes is lethal; and after long exposures, 32 mg/m^3 (10 ppm) is lethal.[41] Three responses to acute exposure characteristic of arsine poisoning are abdominal pain, dark-red urine, and jaundice.[43] Dark-red urine usually appears four to six hours after inhalation of high levels of the gas and is due to a breaking of red blood cells. This is followed at 24 to 48 hours by the appearance of jaundice. Death, if it occurs, is usually due to kidney failure caused by the breaking of red blood cells and the accumulation of hemoglobin in the kidneys.[40] Stibine exposure produces acute responses and has acutely toxic concentrations similar to those of arsine, according to early animal experiments.[43] Human exposure data consist of reports of occupational exposure to arsine: only one case of stibine exposure was located, and that involved combined exposure to arsine, stibine, and hydrogen sulfide.[44] Treatment for severe acute arsine poisoning consists of (1) exchange transfusions to replace red blood cells, and (2) dialysis, if renal failure occurs.[42,45-48]

5.3.3 Chronic Toxicity

The issue more relevant to persons in contact with lead-acid batteries and the gases released during charging is that of responses to low levels of

stibine or arsine. Data on low-level responses to stibine are not available. In the case of arsine, C.A. Nau found a decrease in red blood cell count and hemoglobin levels to near 80% of normal in guinea pigs exposed to arsine for several hours daily at concentrations of from 10 to 40 times the OSHA standard, i.e., from 2 mg/m^3 to 8 mg/m^3.[49] Other blood cell changes were mentioned in that report, but were presented in a descriptive way with no quantitative information.

Two articles report human responses to low levels of arsine.[50,51] In one case, slight decreases in hemoglobin levels were observed in five men exposed for six days to an atmosphere containing arsine.[50] The five men had no symptoms that were common to all, but headache and shortness of breath were listed as symptoms present in four and three of the five cases, respectively. Men exposed to this atmosphere for eight months were not acutely ill; blood analyses showed a striking reduction of hemoglobin and red blood cell levels, however, and blood transfusions were performed. Responses to low levels of arsine were thus found to be cumulative with time. Symptoms common among the men exposed for six months were shortness of breath on exertion, general malaise, nausea, poor appetite, palpitation on exertion, and headache.

In the second report, hemoglobin levels in a group of workers exposed to low levels of arsine at a zinc smelter gradually rose from 85% to 90% over a period of 40 weeks following installation of positive exhaust ventilation.[51] The general plant population had hemoglobin levels of 92%. Arsine concentrations inside the leaching tanks responsible for arsine generation in the plant were 2 to 3 ppm, with no arsine detectable outside the tanks. It was concluded that chronic exposure to arsine levels as low as 0.05 ppm may result in reduced hemoglobin levels. Further, return to normal hemoglobin levels is slow, even after elimination of exposure.

Information on human exposure to low levels of stibine is not available, and the toxicity of stibine must be inferred by its analogy to arsine.

5.4 HEALTH EFFECTS OF ANTIMONY TRIOXIDE

Antimony trioxide (Sb_2O_3) is generated during and after charging of lead/acid EV batteries containing antimony because of decomposition of the stibine produced. At the end of a regular charge, Sb_2O_3 concentrations of 2.4 mg/m^3 and 1.2 mg/m^3 are projected for the inside of a 100-m^3 garage and a 5.5-m^3 vehicle, respectively (Sec. 3.1.1), based on calculations using recent measurements of stibine production during charging.[36] These concentrations exceed the OHSA standard for exposure to Sb_2O_3 (0.5 mg/m^3) by factors of 3 to 5 and indicate a need for garage and vehicle ventilation during charging. An average concentration in a closed vehicle of 0.30 mg Sb_2O_3/m^3 is estimated for a 40-minute trip during discharge (Sec. 3.1.1). This concentration is also close to the OSHA standard concentration and indicates a potential need for ventilation and for careful analysis of the documentation for the OSHA standard.

5.4.1 Metabolism

Sb_2O_3 is eliminated from mice and dogs with a fairly short half-life (40-45 days) following inhalation exposure.[52,53] It is translocated from the lung, principally to liver and skeleton. It deposits in high concentrations in the thryoid. Excretion occurs through both urine and feces, with the urinary route predominating after termination of exposure to dietary Sb_2O_3.[54]

5.4.2 Acute Toxicity

Unlike stibine (SbH_3), inhaled Sb_2O_3 is not acutely toxic, producing no deaths in rats following a four-hour exposure to 2760 mg/m^3.[55] Acute responses noted are skin and eye irritation.

5.4.3 Chronic Toxicity

Recently chronic exposure of rats to Sb_2O_3 at 4.2 mg/m^3 for one year produced lung tumors in 15 of 17 exposed rats; at 1.6 mg/m^3, no lung tumors were found in 14 exposed rats.[56] A NIOSH study has since confirmed the findings of lung tumors following exposure to Sb_2O_3.[57] Other lifetime studies

analyzing causes of death following exposure of animals to relatively low levels of Sb_2O_3 have not been conducted. Toxic responses in animals chronically exposed via inhalation to higher levels of Sb_2O_3 (45-125 mg/m^3) include changes in lungs, liver, spleen, and blood cells.[58, 59] Among persons occupationally exposed to antimony, dermatitis, gastrointestinal irritation, and pneumoconiosis are reported,[60-64] with pneumoconiosis incidences among workers of 10 to 15%.[65,66] Both positive and negative findings are reported for increased lung cancer deaths among antimony smelter workers.[67] The numbers of exposed workers in all cases are small, making conclusions uncertain.

If the lead/acid battery with an antimony electrode additive is to be commericialized for EV use, additional measurements of antimony emissions need to be obtained. The effects of daily exposure of animals to Sb_2O_3 at levels near the OSHA standard should be examined. In addition to the carcinogenic effects mentioned, responses of children to antimony deposition in the thyroid following in-garage exposure to Sb_2O_3 could be a point of concern.

References for Section 5

1. C.D. Brown and J.R.B. Curtiss. "Quantification of Nonlethal Health Risks from Energy Tehcnologies: The Level of Risk Posed by Exposure to Metals in the Environment" (in preparation), 1980.

2. M.R. Moore et al. "Lead," In: "Environment and Man, Vol. 6, The Chemical Environment," Lenihan and W.W. Fletcher, Eds., Academic Press, New York, 1977.

3. J.L. Smith. "Metabolism and Toxicity of Lead" in "Trace Elements in Human Health and Disease, Vol. II Essential and Toxic Elements," A.S. Prasad, and D. Okerlas, Eds., Academic Press, New York, 1976.

4. P.H. Granjean. "Widening the Perspectives of Lead Toxicity," Environ. Res. 17:303-321, 1978.

5. R.A. Goyer and B.C. Rhyne. "Pathological Effects of Lead," In: "International Review of Experimental Pathology," Vol. 12:1-76, G.W. Richter and M.A. Epstein, Eds., Academic Press, New York. 1973.

6. S. Hernberg. "Biochemical, Subclinical and Clinical Responses to Lead," in: "Effects and Dose Response Relationships of Toxic Metals," G.F. Nordberg, Ed. Elseiver, Amsterdam, 1976.

7. O. Wada. "Human esponses to Lead and Their Background with Special Reference to Porphyrin Metabolism" in "Effects and Dose-Response relationships to Toxic Metals." G.F. Nordberg, Ed., Elsevier, Amsterdam, 1976.

8. World Health Organization, "Lead, Environmental Criteria 3," WHO, Geneva, 1977.

9. S. Sassa et al. "Studies in Lead Poisoning," Biochemical Medicine, 8:135-148, 1973.

10. J.R. Reigart and N.H. Whitlock. "Longitudinal Observations of the Relationship between Free Erythrocyte Porphyrins and Whole Blood Lead," Pediatrics 57:54, 1976.

11. A.M. Lee and J.F. Fraumeni. "Arsenic and Respiratory Cancer in Man: An Occupational Study," JNCI 42(6):1045-1052, 1969.

12. S.S. Pinto et al. "Mortality Experience in Relation to a Measured Arsenic Trioxide Exposure," Environ. Health Perspect. 17:127-130, 1977.

13. M.G. Ott et al. "Respiratory Cancer and Occupational Exposure," Arch. Environ. Health 29:250-255, 1974.

14. F. Friberg et al. "Cadmium in the Environment," 2nd ed., CRC Press. Cleveland, OH, 1974.

15. R.K. Sharma et al. "Ecological and Biomedical Effects of Effluents from Near-Term Electric Vehicle Storage Batteries," ANL/ES-90, Argonne National Laboratory, Argonne, IL, 1980.

16. H.D. Livingston. "Measurement and Distribution of Zinc, Cadmium, and Mercury in Human Kidney Tissue," Clinical Chemistry 18:67-72, 1972.

17. M. Piscator. "Cadmium-Zinc Interactions," in: Proc. Internat'l. Symposium on Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Luxemborg, Commission of the European Communities, pp. 951-959, 1975.

18. M. Piscator and B. Lind. "Cadmium, Zinc, Copper, and Lead in Human Renal Cortex," Arch. Environ. Health 24:426-431, 1975.

19. J. Parizek. "Interrelationships among Trace Elements," in: "Effects and Dose-Response Relationships of Toxic Metals," G.F. Nordberg, Ed., Amsterdam: Elsevier Scientific Publishing Co., pp. 498-510, 1976.

20. C.G. Elinder, M. Piscator, and L. Linnman. "Cadmium and Zinc Relationships in Kidney Cortex, Liver, and Pancreas," Environ. Res. 13:432-440, 1977.

21. M. Nordberg, C.G. Elinder, and B. Rahnster. "Cadmium, Zinc and Copper in Horse Metallothionein," Environ. Res. 20:341-350, 1979.

22. L. Friberg et al. "Cadmium, Handbook on the Toxicology of Metals," L. Friberg, G.F. Nordberg and V.B. Vouk, Eds., Amsterdam: Elsevier/ North Holland Biomedical Press, 1979.

23. P.A. Peterson, P.E. Evrin and I. Berggard. "Differentiation of Glomerular, Tubular, and Normal Proteinuria: Determinations of Urinary Excretion of β_2-Microglobulin, Albumin, and Total Protein," J. Clin. Invest. 48:1189-1198, 1969.

24. M. Piscator. "Proteinuria in Chronic Cadmium Poisoning. I. An Electrophoretic and Chemical Study of Urinary and Serum Proteins from Workers with Chronic Cadmium Poisoning," Arch. Environ. Health 4:607-621, 1962.

25. T. Kjellstrom. "Epidemiological Evaluation of Proteinuria in Long-Term Cadmium Exposure with Discussion of Dose-Response Relationships," in: "Effects and Dose-Response Relationships of Toxic Metals," G.F. Nordberg, Ed., 1976.

26. A.J. Pesce and M.R. First. "Proteinuria: An Integrated Review," Marcel Dekker, Inc., New York, 1979.

27. H. Roels et al. "Critical Concentration of Cadmium in Renal Cortex and Urine," Lancet I:221, 1979.

28. K. Nogawa, A. Ishizaki, and S. Kawano. "Statistical Observations of the Dose-Response Relationships of Cadmium Based on Epidemiological Studies in the Kakeshashi River Basin." Environ. Res. 15:185-198, 1978.

29. K. Nogawa, A. Ishizaki, and E. Kobayashi. "A Comparison between Health Effects of Cadmium and Cadmium Concentration in Urine among Inhabitants of the Itai-itai Disease Endemic District," Environ. Res. 18:397-409, 1979a.

30. K. Nogawa, E. Kobayashi, and R. Honda. "A Study of the Relationship between Cadmium Concentrations in Urine and Renal Effects of Cadmium," Enviorn. Health Perspectives 28:161-168, 1979.

31. K. Ellis. "Dose Effects Studies in Cadmium Smelter Workers," 2nd Annual Park City Environmental Health Conference, March 27-29, 1980.

32. United States Environmental Protection Agency, EPA, "Health Assessment Document for Cadmium," (preprint), EPA-600/8-79-003, USEPA, Environmental Criteria and Assessment Office, Research Triangle Park, NC, 1979.

33. G. Kazantzis. "Renal Rubular Dysfunction and Abnormalities of Calcium Metabolism in Cadmium Workers," Environ. Health Perspectives 28:155-159, 1979.

34. T. Kjellstrom, P.E. Evrin and B. Rahnster. "Dose-Response Analysis of Cadmium Induced Tubular Proteinuria. A Study of Urinary β_2-Microglobulin Excretion Among Workers in a Battery Factory," Environ. Res. 13:303-317, 1977b.

35. "Handbook of Labor Statistics (1977)," U.S. Department of Labor, Bureau of Labor Statistics, Bulletin No. 1966.

36. R.O. Loutfy, E.R. Hayes, D.G. Graczyk, R. Varma, F.L. Williams, and N.P. Yao. "Stibine/Arsine Monitoring during Electric Vehicle Operation: Preliminary Test at ANL, Interim Report No. 1," Chemical Engineering Division, Argonne National Laboratory, 1980.

37. National Institute for Occupational Safety and Health. "Criteria for a Recommended Standard . . . Occupational Exposure to Inorganic Arsenic," Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH), Publication No. 75-149, 1975.

38. R.E. Smith et al. "The Tissue Distribution of Radioantimony Inhaled as Stibine," J. Lab. Clin. Med. 33:635-643, 1948.

39. G.A. Levvy. "A Study of Arsine Poisoning," J. Exp. Physiol. Med. Sci. 34:47-67, 1947.

40. B.A. Fowler and J.B. Weissberg. "Arsine Poisoning," New Eng. J. Med. 291:1171-1174, 1974.

41. U.S. Department of Health, Education and Welfare. "Arsine (Arsenic Hydride) Poisoning in the Workplace," NIOSH Current Intelligence Bulletin #32, August 3, 1979.

42. R. Muehreke and C.L. Pirani. "Arsine-Induced Anuria: A Correlative Clinicopathological Study with Electron Microscope Observations," Ann. Intern. Med. 68:853-866, 1968.

43. A. Stock and O. Guttman. "Uber den Antimonwasserstoff und das Gelbe," Antimon. Ber. Dtsch. Chem. Ges 37:885-900, 1904.

44. C.A. Nau, W. Anderson, and R.E. Cone. "Arsine, Stibine, and Hydrogen Sulfide--Accidental Industrial Poisoning by a Mixture," Ind. Med. 13:308-310, 1944.

45. A.E. DePalma. "Arsine Intoxication in a Chemical Plant: Report of Three Cases," J. Occup. Med. 11:582-587, 1969.

46. R. Neuwirtova et al. "Acute Renal Failure Following an Occupational Intoxication with Arsine (AsH_3) Treated by the Artifical Kidney," Act. Med. Scand. 170:535-546, 1961.

47. B. Nielson. "Arsine Poisoning in Metal Refining Plant: Fourteen Simultaneous Cases," Acta Med. Scand. (Suppl.) 496:1-31, 1968.

48. S.P. Wilkinson et al. "Arsine Toxicity Aboard the Asia-Freighter," Br. Med. J. 3:559-563, 1975.

49. C.A. Nau. "The Accidental Generation of Arsine Gas in an Industry," Southern Med. J. 41:341-344, 1948.

50. F.M.R. Bulmer et al. "Chronic Arsine Poisoning among Workers Employed in the Cyanide Extraction of Gold: A Report of Fourteen Cases," J. Ind. Hyg. Toxicol. 22:111-124, 1940.

51. G.A. Johnson. "An Arsine Problem," Am. Ind. Hyg. Qt. 14:188-190, 1953.

52. R.G. Thomas et al. "Retention Patterns of Antimony in Mice Following Inhalation of Particles formed at Different Temperatures," Proc. Soc. Exp. Biol. Med. 144:544-550, 1973.

53. S.W. Felicetti, R.G. Thomas, and R.O. McClennan. "Retention of Inhaled Antimony-124 in the Beagle Dog as a Function of Temperature of Aerosol Formation," Health Phys. 26:525-531, 1974.

54. P. Gross et al. "Toxicological Study of Calcium Halophophate Phosphors and Antimony Trioxide. I. Acute and Chronic Toxicity and some Pharmacologic Aspects," AMA Arch. Ind. Health Occup. Med. 11:473-478, 1955.

55. Industrial Bio-test Laboratories, Inc. Reports, November, December, 1972, IBT Nos. T2298, A2297.

56. W.D. Watt, E.V. Perrin, and E.J. Kerfoot. "Chronic Inhalation Toxicity of Antimony Trioxide," 20th Annual American Industrial Hygiene Conference, Houston, TX, May 18-23, 1980.

57. Personal Communication, W.D. Watt, Wayne State University, Department of Occupational and Environmental Health, Detroit, MI, 1980.

58. C.U. Dernehl, C.A. Nau, and H.H. Sweets. "Animal Studies on the Toxicity of Inhaled Antimony Trioxide," J. Ind. Hyg. Toxicol. 27:256-262, 1945.

59. P. Gross et al. "Toxicologic Study of Calcium Halophosphate Phosphors and Antimony Trioxide. II. Pulmonary Studies," AMA arch. Ind. Health Occup. Med. 11:479-486, 1955.

60. P. Schrumf and B. Zabel. "Clinical and Experimental Studies on Antimony Poisoning of Type Setters," Arch. Exp. Pathol. Pharmakol. 63:242-254, 1910.

61. A.B. Selisky. "Industrial Skin Diseases due to Antimony Salts in the Textile Industry," Dermatol. Wochenschr. 86:723-727, 1928.

62. A Feil. "The Role of Antimony in Industrial Pathology," Presse Med. 47:1133-1134, 1939.

63. L.E. Renes. "Antimony Poisoning in Industry," AMA Arch. Ind. Hyg. 7:99-108, 1953.

64. T. Oliver. "The Health of Antimony Oxide Workers," Brit. Med. J. 1:1094-1095, 1933.

65. "Literature Study of Selected Potential Environmental Contaminants--Antimony and its Compounds," pp. 84-89, Arthur D. Little, Inc., Report for U.S. Environmental Protection Agency, EPA-560/2-76-002, 1976.

66. D.A. Cooper, et al. "Pneumoconiosis among Workers in an Antimony Industry," Am. J. Roentgenol. Radium Ther. Nucl. Med. 103:495-508, 1968.

67. "Supplemental Documentations of the Threshold Limit Values for Substances in Workroom Air," American Conference on Governmental Industrial Hygienists Threshold Limit Committee, p. 437-438, 1977.

6. PERSPECTIVE ON RESULTS

6.1 HUMAN HEALTH

6.1.1 Major Identifiable Effects

Effects of environmental exposure to specified levels of lead, arsenic, and cadmium in air are presented. Projected effects are based on dose-response models for these elements developed from the toxicological and epidemiological literature.

Effects of exposure to airborne lead at distances 5, 15, and 30 km from a mine-mill complex, a primary smelter, and a secondary smelter-battery manufacturing complex are reported. An individual's probability of developing a blood lead concentration greater than 60 μg/dl, a level indicative of increased potential for central nervous system responses to lead, ranges from 0.0013 to 0.0032, depending on distance from the industrial site (Table 5.1). Upper 95% confidence limits for these estimates indicate an enhanced potential for adverse responses to lead over background levels at distances 5 to 15 km from the sites analyzed. Individual risk corresponding to background levels is found at 50 km from the mine-mill complex. Effects due to dispersal of lead through air at great distances from the site are therefore not anticipated. Significant increases in battery industries are projected by the year 2000 (Table 3.2); increasing numbers of persons could therefore be exposed to lead and experience excess risk of developing central nervous system responses.

In the case of arsenic, excess person-years lost as a result of increased lung cancer mortality rates in the vicinity of a secondary lead smelter-battery manufacturing complex range from 0.23 excess person-years lost (about 84 days) at 5 km to 0.02 excess person-years lost at 30 km from the source

(Table 5.2). These values are based on a group of 10,000 persons examined over a ten-year period following 30 years of exposure. The analysis suggests that the increase in risk is negligible from environmental arsenic exposure due to secondary smelting of lead required for increased lead/acid EV battery production.

In the case of cadmium, an increased excess risk of developing renal tubular proteinuria is indicated with increasing cadmium levels associated both by proximity to a lead mine-mill complex and accumulation in the body over time (Table 5.3). At the 5-km distance there is a considerable increase in risk after ten years (1-3 per 1000). After a period of 30 years, the excess risk is about 2 per 100, which would imply a significant level of risk. A considerable degree of risk, approximately 2 per 1000, is experienced by those persons at the farthest distance only after a period of 30 years. For those at the middle distances, risk also is most noteworthy at the 30-year point (3-4 per 1000), and is moderate (2-4 per 10,000) at the ten-year point. The excess risk becomes insignificant (defined as 1 per 10^6) at 85 km from the site. Effects due to dispersal of cadmium through air at great distances from the site are therefore not anticipated. Significant increases in mining and milling of lead anticipated by the year 2000 (Table 3.2) could result in focal areas of increased exposure to cadmium with accompanying excess risk of developing renal tubular proteinuria.

Health effects among the occupationally exposed are also assessed using the toxicological models. When possible occupational exposure levels were used in conjunction with nonoccupational exposures in the dose-response functions, a range of results was seen. The likelihood of having a blood-lead level defined as hazardous was 1 per 100. In a cohort of 10,000 persons followed for ten years (100,000 person-years), 53 person-years could be expected to be lost due to arsenic exposure. Finally, the excess risk of cadmium-induced tubular proteinuria could be about 0.325, using a very conservative definition of the disease.

In an analysis of occupational safety, mean occupational injury and illness rates for persons in the following industries are substantially higher than those reported for persons employed both in other industries related to

those required for EV production and in the entire private sector: battery breaking, secondary lead smelting and refining, storage battery manufacture, and recycling industries for nickel and cobalt (Tables 5.5 and Appendix H, Tables H.2 and H.4). Mean occupational injury and illness rates in the latter battery-related industries range from 23 to 28 cases per 100 full-time workers, compared to rates of 11 to 17 in the related industry category and 9 in the entire private sector. Increased employment in these latter industries for the purpose of increased near-term EV manufacture (Tables 3.2, 4.5, and 4.6) could result in a population of persons experiencing a mean rate of occupational injury and illness 1.5- to 2-fold higher than persons with similar skills employed in related industries and approximately 3-fold higher than the mean for the entire private sector.

Acute and chronic effects of exposure to stibine, arsine, and antimony trioxide are described. A potential for reduction in blood hemoglobin levels is identified following chronic low-level exposure to stibine or arsine. Pneumoconiosis with a potential for development of lung tumors is identified as an effect of chronic low-level exposure to antimony trioxide.

The Health Effects section of this document has concentrated on the presentation of a methodology hitherto unused in studies of this type, i.e., metabolic dose-response models with lethal or nonlethal endpoints. This method of risk projection appears to be well suited to general population exposure levels. The models presented do not exclude the use of other endpoints in future analyses. Given that the approach presented receives support in terms of fulfilling expectations for the HEED program, the methodology can be expanded to include other elements and other toxicological endpoints, as well as additional exposure routes. Models used in the present analysis also can be refined and improved to provide a more complete analysis of human health effects.

6.1.2 Major Uncertainties

The following four major uncertainties are associated with the human health risk analysis that is based on dose-response modeling: (1) uncertainties in the cause-effect relationships (in animals, cause-effect relationships

are established through experimentation; in humans, cause-effect relationships are established with less certainty via epidemiological studies); (2) statistical uncertainties always associated with epidemiological studies due to shortcomings in the data base; (3) uncertainties due to assumptions concerning the uptake, distribution, and retention of the compounds analyzed in order to arrive at body burdens corresponding to given levels of exposure; and (4) uncertainties due to variations in susceptibility among persons in the exposed populations. The approach taken was to deal with these uncertainties and arrive at a dose-response model based on the best information available.

In the analysis of occupational health and safety impacts, the grouping of industries together in reporting occupational injury and illness rates limited the certainty with which a given industry could be assigned a given incidence rate. This limitation was dealt with by clearly indicating the SIC code number and industry description used to assign a given rate to a given industry required for EV battery production. The uncertainties due to grouping could then be analyzed by the reader. In addition, the analysis assumes a stable occupational injury and illness rate for a given type of industry. Predictions of occupational safety impacts could be in error if new industries differ substantially from those presently operating in terms of injury rates among workers.

The limited number of animal experiments assessing the effects of exposure to low levels of arsine and the complete lack of data on low-level effects of stibine limit the depth of present knowledge and the confidence one can have in the standards set for these compounds. The carcinogenic potential of antimony trioxide needs to be further investigated to validate recent findings.

6.1.3 Major Policy Issues

If the OSHA standard for inorganic arsenic and arsine were decreased from 0.2 mg/m^3 to 0.002 mg/m^3, as recommended in a 1975 NIOSH criteria document, concentrations of arsine produced during charging of lead/acid EV batteries would exceed the new OSHA limit.

Concentrations of antimony trioxide in the vehicle and the garage at the end of a regular charge are calculated to be three to five times greater than the OSHA standard (0.5 mg/m^3). Measurements of antimony trioxide buildup following stibine release thus need to be made. Recent reports on the induction of lung tumors in rats by antimony trioxide need to be further investigated.

6.2 ECOSYSTEM EFFECTS

6.2.1. Major Identifiable Effects

Lead/acid, nickel/zinc, and nickel/iron battery-related emissions could cause adverse environmental impacts within several kilometers of battery-related industries. Projected concentrations of lead, antimony, copper, zinc, nickel, cadmium, arsenic and sulfur exceed estimated permissible concentrations (EPCs) in air, soil, or water near one or more of the battery-related industries.

Environmental cycling of emission constituents will be highly site- and species-specific. High concentrations of these constituents can retard germination and inhibit nutrient recycling resulting in disruption and possible elimination of ecosystem structure and function. Bioconcentration of these elements above ambient levels is likely in aquatic ecosystems but not in terrestrial ecosystems, although in terrestrial ecosystems higher-than-normal tissue concentrations could occur in areas contaminated with these elements. Bioconcentration of potentially toxic elements can result in acute and chronic toxicity to exposed biota. Biomagnification of elements along the food chain leading to man is not likely to be an important factor caused by major emission constituents from the battery cycles.

Groundwater supplies could be adversely affected, both quantitatively and qualitatively, if aquifers are intersected during mining operations. Physical surface disturbances resulting in habitat distruction also will occur as battery-related industries are established. Land reclamation should mitigate effects associated with surface disturbances. Long-term land allocation will be required for many battery-related industries.

6.2.2 Major Uncertainties

Quantification of environmental impacts from the lead/acid, nickel/zinc, and nickel/iron battery cycles is limited by a paucity of information relating environmental responses to emission levels and an inherent imprecision of our knowledge about emission levels. Future control technologies and regulations, future battery technologies, and actual levels of emissions are uncertain.

APPENDIX A. DETAILED DESCRIPTION OF THE DOSE-RESPONSE MODELS PRESENTED IN SECTION 5

A.1 LEAD

The initial step in the lead analysis is to relate an ambient airborne lead concentration (Pb-A) experienced by an exposed population to an instantaneous blood lead concentration ($Pb\text{-}B_i$). A mathematical relationship for this association can be derived from a study by Azar,[1] who measured $Pb\text{-}B_i$ in sample adult groups from various cities of the United States. The data from this study demonstrate that $Pb\text{-}B_i$ correlates with ambient Pb-A by the following functional relationship:

$$\log (Pb\text{-}B_i) = 1.226 + 0.153 \log (Pb\text{-}A) , \quad (1)$$

where Pb-A is the ambient air lead concentration in units of $\mu g/m^3$ and $Pb\text{-}B_i$ is the instantaneous blood lead concentration in units of µg/100 ml of whole blood.

Using data from various studies[2-4] a functional relationship between $Pb\text{-}B_i$ and mean erythrocyte protoporphyrin concentration (EP) can then be derived as follows:

$$\overline{EP} = 32.44\ e^{0.031(Pb\text{-}B_i)} , \quad (2)$$

where $\overline{EP}$ is given in units of µg/100 ml of whole blood.

In Equation 2, the association of EP response to $Pb\text{-}B_i$ gives a mathematical relationship between instantaneous blood lead concentration and a long-term subclinical response (EP). The estimated average value, $\overline{EP}$, can

then be interpreted in terms of an overall measure of physiological response to a given lead exposure.

If selection is made of a unique value for EP that is indicative of an elevated response, not necessarily an adverse response, and in addition, if it is assumed that the underlying statistical distribution of EP values in an exposed population is normal, then it is possible to calculate a standard normal deviate with a specific probability for unique EP values as:

$$\frac{EP - \overline{EP}}{S_{\overline{EP}}} \equiv P(EP) \ , \qquad (3)$$

where the standard error ($S_{\overline{EP}}$) used in the denominator of Equation 3 is derived from the data set used in the derivation of Equation 2. The test statistic can, in turn, be related to a distinct probability P(EP) or measured likelihood for EP values to be at or above the defined value.

The choice of the cutoff EP value used in the present analysis was directed by the availability of data in the literature. The value 82 µg/100 ml was selected from the work of Roels,[3] where he defined a value of EP indicative of increased lead absorption. The result of Equation 3, P(EP) can be interpreted as a prevalence for individual EP responses at or in excess of 82 µg/100 ml and therefore P(EP ≥ 82) is the likelihood of observing a subclinical physiological response (EP) in a defined nonoccupational population under long-term exposure to lead.

Use of this association makes possible the calculation of an associative relationship between the prevalence value for an elevated EP and a continuous blood lead concentration ($Pb\text{-}B_c$) as follows:

$$\overline{Pb\text{-}B_c} = \exp\left(\frac{P' + 14.75}{4.89}\right) . \qquad (4)$$

In this relationship P' is a transform of the following:

$$P' = \ln\left(\frac{P}{100 - P}\right) ,$$

where P is the P(EP) prevalence value calculated in Equation 3. The relationship thus gives a measure of mean blood lead concentration for a continuously exposed population.

The remaining step in the risk estimation is to relate a specific $Pb\text{-}B_c$ value to a specific lead-induced disease state. This is accomplished by following a procedure similar to that used in step 3. In this case the mean $Pb\text{-}B_c$ value and a $Pb\text{-}B_h$ value (a blood lead concentration defined as hazardous, in this case $Pb\text{-}B_h$ = 60 µg/100) are used to generate a measure of likelihood of the presence of a lead exposure defined as hazardous:

$$\frac{\ln (Pb\text{-}B_h) - \ln \overline{(PB\text{-}B_c)}}{\ln \left(S_{\overline{Pb\text{-}B_c}} \right)} = P(Pb\text{-}B_c \geq Pb\text{-}B_h) \ . \qquad (5)$$

In this relationship we use the observation that Pb-B tends to a log normal distribution; therefore, the standard normal deviate for a log-transformed value of $Pb\text{-}B_c$ can be interpreted as a measure of likelihood of population risk. For the present situation, we have chosen a $Pb\text{-}B_h$ value of 60 µg/100 ml, a value indicative of adverse central nervous system response.[5] This estimate of likelihood of having a $Pb\text{-}B_c \geq$ 60 µg/100 ml then forms the estimate of overall population risk from a given exposure.

As a first approximation, the confidence limits based on the 95 percent confidence bounds of Equation 2 can be used to bound the overall measure of risk. In this case,

$$\pm\ 95\%\ \overline{EP} = 1.96\ SE_{\overline{EP}} + \overline{EP} \ , \qquad (6)$$

where the bounds are generated by using these limiting values in the calculational steps from 3 to 5 to determine likelihood estimates for the calculated given bounding values for the lead model.

A.2 ARSENIC

The exposure dependency relationship for arsenic was derived from the data published by Ott[6] and Pinto.[7] Exposure level estimates were recalculated from published estimates. In Section 5.1.3 it was noted that the general form of the equation is

$$y = e^{a} x^{b} \tag{7}$$

where y is the standardized mortality ratio for respiratory cancer and x is the annual mean exposure to arsenic trioxide in $\mu g/m^3$.

The coefficients of Equation 7 were calculated to be a = -0.22; b = 0.35, and the actual relationship was determined to be:

$$SMR = 0.801(As-A)^{0.35} . \tag{8}$$

If we use the basic definition of the SMR as the ratio of the mortality rate of a population under some defined exposure situation (MR_e) to that observed for a reference, population (MR_r), it is possible to solve for the mortality rate of the exposed group in terms of the ambient air concentration so that we have:

$$MR_e = 0.801(MR_r)(As-A)^{0.35} . \tag{9}$$

This function can then be used to estimate a level of response in an exposed cohort if we define the mortality rate of Equation 9 as the cohort mortality rate and follow the group through ten years of life experience at the estimated level of response. The difference between the response magnitudes of the exposed and reference cohort will form the basis for an estimate of impact from the defined exposure level.

Equation 10 represents the case in which an exponential decay process is assumed:

$$n_k = n_o e^{-\beta \Delta t} \tag{10}$$

In such a situation, n_o, the cohort size at some initial time (t_o), is reduced to n_k over the time interval $\Delta t = t_k - t_o$. This relationship depends on the parameter β, which for any single year can be interpreted as numerically equivalent to MR. In Equation 11 below, β_1 and β_2 represent responses to two different exposure levels, as in a reference and exposed situation. Integrating under the curves for a time interval Δt gives the number of person years lived for each exposure. The difference between these areas is a measure of the relative mortality experience, excess person years lost, for the exposed cohort. This relative measure can be expressed as:

$$\text{excess person years lost} = n_o\left[\frac{1}{\beta_1}\left(1 - \exp(-\beta_1 t_k)\right) - \frac{1}{\beta_2}\left(1 - \exp(-\beta_2 t_k)\right)\right], \quad (11)$$

where n_o is the initial cohort size, t_k is end point of the time interval, and β_1 and β_2 are defined as above.

The long-distance transport of arsenic released by activities in an electric storage battery industry has been questioned as a possible source of general population health impact. Analysis of this issue shows that such long-range transport is not a significant concern relative to the overall population response to toxic emissions from the battery industry.

Three components of the battery industry with measurable arsenic emissions (E) are identified in Table 3.1 of this report. They are primary and secondary smelting of lead (870 and 320 g As/day) and battery manufacture (2.6 g As/day). The following is a brief discussion of the approach and assumptions used to evaluate the health implications of these emissions.

1. A single exposure effect coefficient, R, was calculated from Equation 8 for an exposure of 1 $\mu g/m^3$ of arsenic. This coefficient was determined to be 5.31×10^{-5} deaths/person/$\mu g/m^3$.

2. A point source emission equivalent to the industrial arsenic release is located on the West Coast of the continental United States and is allowed to disperse in an easterly direction to the Eastern Seaboard. A constant wind velocity of 5 m/s and an exponential decay of 1% of the mass per hour is assumed.

3. A population density of 22.9 persons/km^2 (equivalent to the average density of the United States) under the dispersed emission is assumed.

4. Health response as death from cancer of the lung, bronchus, and trachea represents the population response to airborne arsenic. Unit population exposure per hour was summed over the total number of hours equivalent to the time of transit from west to east, and the number of total deaths from lung cancer was used to describe an annual national response.

A.3 CADMIUM

In the cadmium dose-response model, a daily dose can be derived, based on ambient air levels and several assumptions regarding food intake and absorption rates via either pathway.

Assuming a constant dose level for an annual exposure, the total cadmium body burden (B_i) at the end of one year can be calculated with the use of a decay function:[6]

$$B_i = a_i \left(\frac{1 - k^{365}}{1 - k} \right), \tag{12}$$

where

a_i = daily dose, and
k = fraction of the body burden remaining at the end of one day.

With expansion of the constant dose level assumption to a period of years, the total body burden at the end of n annual intervals can be derived by summing the product of B_1 and a factor which accounts for further decay. The form of this summation would be:

$$B_n = \sum_{i=1}^{n} B_1 (k^{365})^{n-i} . \tag{13}$$

The kidney cortex concentration is estimated by assuming a third of the total body burden resides in the kidneys and the cortex levels are 50% greater than the mean concentration for the whole kidney.[8,9] The population mean kidney cadmium concentration can be directly related to a mean β_2-microglobulin level through a functional relationship of the form:

$$\ln \beta_2\text{-MG} = a + b(\text{Cd-K}) \qquad (14)$$

where

ln β_2-MG = the natural logarithm of the β_2-microglobulin concentration in urine (μg/L),

Cd-K = the cadmium concentration in the kidney cortex (μg/g), and

a,b are coefficients derived from the relationship.

This association can be used as the basis for estimates of individual risk by defining a specific β_2-MG response level as indicative of adverse health response. To avoid the possibility of confounding the estimates in the present analysis, all values have been derived from data on persons 50 to 70 years old,[10] the period when the cadmium burden changes the least.

The population mean kidney cadmium concentration can be directly related to a mean β_2-MG level if one assumes that the kidney concentration and log-transformed β_2-MG levels are normally distributed in the population and the two are causally related.[11-14] Having mathematically described the relationship between the mean values, it is possible to solve for a specific value of β_2-MG response (ln β_2-MG/L urine) equivalent to the standard normal deviate for the kidney cortex cadmium concentration. This value can then be related to a table value for the normal probability distribution to derive a level of risk.

A.4 ASSUMPTIONS FOR OCCUPATIONAL APPLICATION OF DOSE-RESPONSE FUNCTIONS

Application of the dose-response functions to the occupational sector is based on the following assumptions:

- Worker exposure is for 8 hours/day, 230 days/year.
- Workers reside 5 km from the industrial complexes and are therefore exposed to the applicable ambient air levels described in Tables 5.1 through 5.3.
- Workers have a more uniform exposure regime than the general public; therefore, the standard errors of the urinary β_2-MG distribution (cadmium model) and the erythrocyte protoporphyrin distribution (lead model) are one-tenth those of the general population.
- For the lead and cadmium model, food and water exposures are the same as those of the respective reference populations.
- During a typical 8-hour work day, 10 m^3 of air is inhaled; the total for the entire 24-hr period is 20 m^3.
- The typical workplace air concentrations used for lead, arsenic, and cadmium were 0.15 mg/m^3, 0.05 mg/m^3, and 0.05 mg/m^3, respectively.

A.5 TABLES SHOWING INDUSTRIAL REQUIREMENTS AND OCCUPATIONAL INJURY AND ILLNESS RATES FOR NICKEL/ZINC AND NICKEL/IRON BATTERY SYSTEMS

Table A.1. Industrial Requirements for Production of Nickel/Zinc Batteries for Electric Vehicle Use

Industry[a]	SIC Code	Output Required[b]		Number of New Production Units Required	
		By Year 2000, MT/day	Per MWh, kg/day/MWh	By Year 2000	Per MWh
Zinc production: mining and milling	1031	540 (Zn)	2.7	4.6[c]	2.3×10^{-5}
primary smelting and refining	3333	540	2.7	1.6[c]	8.0×10^{-6}
Nickel production	3339	780 (Ni)	3.9	2[d]	-
Cobalt production	3339	40 (Co)	0.20	-	-
KOH production	2812	270 (KOH)	1.4	3[e]	1.5×10^{-5}
Polypropylene production	2821	180 (polypropylene)	0.88	0.8[f]	4.0×10^{-6}
Ni/Zn battery manufacture	3691	2600 (batteries)	12.7	-	-
Nickel recycling	3341	42 (Ni)	0.21	-	-
Cobalt recycling	3341	2 (Co)	0.01	-	-

[a] Ni/Zn battery manufacturing, nickel recycling, and cobalt recycling are industries which are necessary for Ni/Zn battery production but which currently do not exist. Output required for nickel and cobalt recycling industries assumes removal of these elements for recycling as indicated in Table 3.5, this report.

[b] Items to be produced are shown in parenthese in the first column. Electric vehicle fleet size by the year 2000 is taken as 8×10^6 vehicles (200,000 MWh), growing at 30%/year (ANL/ES-90, p. 73).

[c] Assumes annual production of 94 MT/day per mine-mill complex (Table 3.6, this report) and 100,000 MT/year per smelter (ANL/ES-90, p. 73).

[d] A large smelter is estimated to produce 140,000 MT/yr (Table 3.6, this report).

[e] Average plant is assumed to produce 35,000 MT/year (ANL/ES-90, p. 78).

[f] Assumes 85,000 MT/year per polypropylene manufacturing unit (ANL/ES-90, p. 35).

Table A.2. Industrial Requirements for Production of Nickel/Iron Batteries for Electric Vehicle Use

Industry[a]	SIC Code	Output Required[b]		Number of New Production Units Required	
		By Year 2000, MT/day	Per MWh, kg/day/MWh	By Year 2000	Per MWh
Steel production: mining steel manufacturing	1011 3312	800 (steel)	4.0	1	5×10^{-6}
Nickel production	3339	490 (Ni)	2.5	1.3	6.5×10^{-6}
Cobalt production	3339	22 (Co)	0.11	-	-
KOH production	2812	220 (KOH)	1.1	2[c]	1×10^{-5}
LiOH production	1479 2819	42 (LiOH)	0.21	5[d]	2.5×10^{-5}
Copper production	1021 3331	110 (Cu)	0.55	-	-
Polypropylene production	2821	250 (polypropylene)	1.3	1[e]	5×10^{-6}
Ni/Fe battery manufacture	3691	3200 (batteries)	16.2	-	-
Nickel recycling	3341	0.52 (Ni)	0.0026	-	-
Cobalt recycling	3341	0.024 (Co)	0.00012	-	-

[a] Ni/Fe battery manufacturing, nickel recycling, and cobalt recycling are industries that are necessary for Ni/Fe battery production but that do not currently exist. Output required for nickel and cobalt recycling industries assumes removal of these elements for recycling as indicated in Table 3.7, this report.

[b] Same as for Table 4.5, footnote b.

[c] Average plant is assumed to produce 35,000 MT/year (ANL/ES-90, p. 101).

[d] Assumes annual production of 5000 short tons/year as lithium carbonates (ANL/ES-90, p. 103).

[e] Assumes 85,000 MT/year per polypropylene manufacturing unit (ANL/ES-90, p. 35).

Table A.3. Occupational Injury and Illness Rates for Industries Involved in Nickel/Zinc Battery Manufacture for Electric Vehicle Use

Industry	SIC Code	Sector	Incidence Rates[a] Total Cases	Lost Workday Cases	Lost Workdays	SIC Industry Description and Code Listing[b]
Zinc production	1031, 3333	SI	6.5, 15.1	4.4, 5.9	69.2, 140.0	Metal mining (10), Primary nonferrous metals (333)
		GRI[b]	11.0, 17.3	5.7, 6.3	113.0, 116.5	Mining, Primary metal industries (33)
		GP[b]	9.1	3.3	56.1	Private sector
Nickel production	3339	SI	20.0	10.1	194.1	Primary nonferrous metals, n.e.c. (3339)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Cobalt production	3339	SI	20.0	10.1	194.1	Primary nonferrous metals, n.e.c. (3339)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
KOH production	2812	SI	7.4	2.1	52.7	Alkalies and chlorine (2812)
		GRI	8.3	2.8	48.2	Chemical and allied products (28)
		GP	9.1	3.3	56.1	Private sector
Polypropylene production	2821	SI	8.8	2.8	43.5	Plastic materials and resins (2821)
		GRI	8.3	2.8	48.2	Chemicals and allied products (28)
		GP	9.1	3.3	56.1	Private sector
Ni/Zn battery manufacture	3691	SI	23.5	10.0	176.0	Storage batteries (3691)
		GRI	11.0	3.8	66.6	Misc. electrical equipment and supplies (369)
		GP	9.1	3.3	56.1	Private sector
Nickel recycling	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Cobalt recycling	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector

[a] Incidence rates represent the number of injuries and illnesses and lost workdays per 100 full-time workers in 1975, as calculated in U.S. Department of Labor, Bureau of Labor Statistics, 1978, Bulletin 1981.

[b] SI, GRI, and GP refer to specific industry, general related industry, and general population, respectively. The SIC industry description and code number used for these industry definitions are shown in the last column of the table.

Table A.4. Occupational Injury and Illness Rates for Industries Involved in Nickel/Iron Battery Manufacture for Electric Vehicle Use

Industry	SIC Code	Incidence Rates[a] Sector	Total Cases	Lost Workday Cases	Lost Workdays	SIC Industry Description and Code Listing[b]
Steel production	1011, 3312	SI[b]	6.5, 11.1	4.4, 3.2	69.2, 69.6	Metal mining (10), Blast furnaces and steel mills (3312)
		GRI[b]	11.0, 17.3	5.7, 6.3	113.0, 116.5	Mining, Primary metal industries (33)
		GP[b]	9.1	3.3	56.1	Private sector
Nickel production	3339	SI	20.0	10.1	194.3	Primary nonferrous metals, n.e.c. (3339)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Cobalt production	3339	SI	20.0	10.1	194.3	Primary nonferrous metals, n.e.c. (3339)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
KHO production	2812	SI	7.4	2.1	52.7	Alkalies and chlorine (2812)
		GRI	8.3	2.8	48.2	Chemical and allied products (28)
		GP	9.1	3.3	56.1	Private sector
LiOH production	1479, 2819	SI	6.0, 8.1	3.5, 2.6	45.6, 53.0	Nonmetallic minerals mining (14), Industrial inorganic chemical, n.e.c (2819)
		GRI	11.0, 8.3	5.7, 2.8	113.0, 48.2	Mining, Chemical and and allied products (28)
		GP	9.1	3.3	56.1	Private sector
Copper production	1021, 3331	SI	6.5, 12.5	4.4, 4.6	69.2, 168.9	Metal mining (10), Primary copper (3331)
		GRI	11.0, 17.3	5.7, 6.3	113.0, 116.5	Mining, Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Polypropylene production	2812	SI	8.8	2.8	43.5	Plastic materials and and resins (2821)
		GRI	8.3	2.8	48.2	Chemicals and allied products (28)
		GP	9.1	3.3	56.1	Private sector
Ni/Fe battery manufacture	3691	SI	23.5	10.0	176.0	Storage batteries (3691)
		GRI	11.0	3.8	66.6	Misc. electrical equipment and supplies (369)
		GP	9.1	3.3	56.1	Private sector
Nickel recycling	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector
Cobalt recycling	3341	SI	28.1	11.6	228.4	Secondary nonferrous metals (334)
		GRI	17.3	6.3	116.5	Primary metal industries (33)
		GP	9.1	3.3	56.1	Private sector

[a]Incidence rates represent the number of injuries and illnesses and lost workdays per 100 full-time workers in 1975, as calculated in U.S. Department of Labor, Bureau of Labor Statistics, 1978, Bulletin 1981.

[b]SI, GRI, and GP refer to specific industry, general related industry, and general population, respectively. The SIC industry description and code number used for these industry definitions are shown in the last column of the table.

References for Appendix A

1. Z. Azar et al. "An Epidemiologic Approach to Community Air Lead Exposure Using Personal Air Samplers," in "Environmental Quality and Safety," Suppl. II, T.B. Griffin and J.H. Knelson, Eds., George Thieme Publ., Stuttgart, 1975.

2. S. Sassa et al. "Studies in Lead Poisoning," Biochemical Medicine, 8:135-148, 1973.

3. H. Roels et al. "Impact of Air Pollution by Lead on the Heme Biosynthetic Pathway," Arch. Environ. Health 31:310-317, 1976.

4. R. Lillis et al. "Prevalence of Lead Disease among Secondary Lead Smelter Workers and Biological Indicators of Lead Exposure," Environ. Res. 14:255-285, 1977.

5. World Health Organization. "Early Detection of Health Impairment in Occupational Exposure to Health Hazards," WHO Tech. Report Series 571, 1975.

6. M.G. Ott et al. "Respiratory Cancer and Occupational Exposure," Arch. Environ. Health 29:250-255, 1974.

7. S.S. Pinto et al. "Mortality Experience in Relation to a Measured Arsenic Trioxide Exposure," Environ. Health Perspect. 17:127-130, 1977.

8. F. Friberg et al. "Cadmium in the Environment," 2nd ed., CRC Press. Cleveland, OH, 1974.

9. H.D. Livingston. "Measurement and Distribution of Zinc, Cadmium, and Mercury in Human Kidney Tissue," Clinical Chemistry 18:67-72, 1972.

10. S.B. Gross, D.W. Yeager, and M.S. Middendorf. "Cadmium in Liver, Kidney, and Hair of Humans, Fetal through Old Age," J. Toxicol. and Environ. Health 2:153-167, 1976.

11. M. Piscator and B. Lind. "Cadmium, Zinc, Copper, and Lead in Human Renal Cortex," Arch. Environ. Health 24:426-431, 1975.

12. K. Shiroishi et al. "Urine Analysis for Detection of Cadmium Induced Renal Changes, with Special Reference to β_2-Microglobulin. A Cooperative Study Between Japan and Sweden," Environ. Res. 13:407-424, 1977.

13. T. Kjellstrom, K. Shiroishi, and P.E. Evrin. "Urinary β_2-Microglobulin Excretion among People Exposed to Cadmium in the General Environment. An Epidemiological Study in Cooperation Between Japan and Sweden," Environ. Res. 13:318-344, 1977.

14. T. Kjellstrom, P.E. Evrin, and B. Rahnster. "Dose-Response Analysis of Cadmium Induced Tubular Proteinuria. A Study of Urinary β_2-Microglobulin Excretion Among Workers in a Battery Factory," Environ. Res. 13:303-317, 1977.

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